

A Dissertation on

**PREVALENCE OF SUBCLINICAL HYPOTHYROIDISM
IN PREGNANCY AND ITS MATERNAL AND FETAL
OUTCOMES**

Submitted to

**THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600032**

In partial fulfilment of the Regulations
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M.S. OBSTETRICS AND GYNACOLOGY

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**DEPARTMENT OF OBSTETRICS AND
GYNAECOLOGY**

R.S.R.M. LYING IN HOSPITAL

STANLEY MEDICAL COLLEGE & HOSPITAL

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APRIL 2017

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This is to certify that **Dr.R. MONICA**, Post - Graduate Student (June 2014 to April 2017) in the Department of OBSTETRICS AND GYNACOLOGY, R.S.R.M. LYING IN HOSPITAL, STANLEY MEDICAL COLLEGE, Chennai - 600 013, has done this dissertation on “**PREVALENCE OF SUBCLINICAL HYPOTHYROIDISM IN PREGNANCY AND ITS MATERNAL AND FETAL OUTCOMES**” under my guidance and supervision in partial fulfillment of the regulations laid down by the Tamilnadu Dr. M. G. R. Medical University, Chennai, for M.S. (Obstetrics and Gynaecology), Degree Examination to be held in April 2017.

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DECLARATION

I, **Dr.R.MONICA**, declare that I carried out this work on **“PREVALENCEOF SUBCLINICAL HYPOTHYROIDISM AND ITS MATERNAL AND FETAL OUTCOMES”** has been prepared by me.

This is submitted to the Tamilnadu DR. M. G. R. Medical University, Chennai in partial fulfilment of the rules and regulation for the M. S. Obstetrics and Gynaecology. This has not been submitted previously by me for the award of any degree or diploma from any other University.

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PREVALENCE OF SUBCLINICAL HYPOTHYROIDISM IN PREGNANCY AND ITS MATERNAL AND FETAL OUTCOMES

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INTRODUCTION

Among endocrine disorder in pregnancy thyroid dysfunction is the second most common. Pregnancy increases the demand on maternal thyroid gland. Thyroid dysfunction has an adverse maternal and fetal effects

Among thyroid dysfunction ,hypothyroid has a relatively high prevalence during pregnancy affecting upto 14.3 % of all pregnant women in India. (dhanwal et al)

Hypothyroidism can be clinical/overt and subclinical .

Clinical hypothyroidism defined as elevated serum level of thyroid stimulating hormones (TSH >10 mIU/L)and subnormal free thyroxine level.

Sub clinical hypothyroid defined as enhanced TSH level, usually beyond the upper reference limit and a normal fT4 level.

Hypothyroidism can be primary and central

Primary hypothyroidism mainly caused by a primary abnormality in the thyroid.

Central hypothyroidism-5% due to lack of TSH and its effects. T3,T4,FT4 were done to diagnose central hypothyroidism.

Subclinical Hypothyroidism:

Subclinical hypothyroidism defined as enhanced value of Serum TSH according to the trimester specific reference and normal free T 4.

Incidence of subclinical hypothyroidism is 2.5% in western countries. (vary with ethnicity)

SCH mostly asymptomatic, but there is evidence of autoimmune thyroid disease (positive TPOAbs and or TGantibodies)in 50-60% (12)

Subclinical hypothyroidism was most frequently seen in women delivering very preterm baby. (before 32 weeks).(12)

Women with Subclinical hypothyroidism had a 3 fold increased risk of placental abruption and 2 fold increased risk of preterm labour compared to euthyroid women.(22) Gestational hypertension also more frequently occurred .(23)

Enhanced maternal TSH (high level of normal) without decreased Thyroid hormone is associated with neonatal respiratory distress, miscarriage and preterm delivery (12).

The likelihood of patients diagnosed as hypothyroids during pregnancy to continue to be hypothyroid even after pregnancy depends on the initial TSH value.

The United States Preventive Services Task force reported that nearly almost all patients with an initial TSH >5 mIU/ml developed overt hypothyroidism within 5 years.(33)

PROBABLE CAUSES OF SCH in pregnant women

Chronic/subacute autoimmune thyroiditis (Hashimoto's thyroiditis is the major cause of SCH in high income countries and it increases with age)

Iodine deficiency

Radioactive iodine therapy

Surgical removal of thyroid gland.

Drugs like

lithium,

Amiodarone,

Interferon alpha,

Interlukin 2,

Rifampicin,

Sunitinib and

Thalidomide.

Presence of goitrogens in diet in India

Risk factors:

Type 1 DM

Autoimmune disease

Family history of hypothyroid

Pathogenesis

At molecular level, thyroid hormone has role in placenta development.

In hypothyroidism early faulty development of placenta due to decreased level of thyroid hormones leads to miscarriage, preeclampsia, abruption and preterm labour. Thyroid hormone synthesis require adequate amount of iodine. Severe deficiency of iodine causes hypothyroidism. It found to be associated with decreased intelligence, cretinism and even congenital anomalies of the fetus.

Intra uterine growth restriction and fetal distress are more common in women with subclinical hypothyroidism.

Complications of subclinical hypothyroidism in pregnancy can be prevented if thyroxine treatment is started in 1st trimester (ideally antenatally).

Treatment after 1st trimester will not eliminate already established fetal neurodevelopmental delay, because it is in the first trimester that the fetus depends completely on maternal thyroid hormone for brain development.

Thyroid function tests-should cautiously interpreted during pregnancy due to physiological changes of thyroid. The cutoff values for diagnosing thyroid dysfunction in pregnant women is not the same as non pregnant women. The diagnosis of hypothyroidism is also difficult in pregnancy because many of the symptoms and signs of hypothyroidism resembles pregnancy symptoms.

Severe clinical/overt hypothyroidism rarely becomes pregnant because most of these women will be infertile and they also have higher rates of miscarriages.

There are not enough studies in pregnant women with hypothyroidism to see if early thyroxine supplementation and adequate treatment actually reduces the occurrence of complications. This study was undertaken to identify prevalence maternal and fetal outcomes, find if promptly diagnosed and adequately treated hypothyroid women were able to avert complications.

REVIEW OF LITERATURE

Prevalence of subclinical hypothyroidism in pregnancy * 2014
European Thyroid Association reported - prevalence of subclinical hypothyroid in pregnancy is 2 -2.5% in west. *China-4% *Belgium-6.8%
*North Spain as high as 13.7%.

Endocrine society guidelines studied data from 523 healthy pregnant women with no known history of thyroid disorder. In this group 65 women (12%) had subclinical hypothyroidism.

Liang Miao chen et al 2014 conducted prospective study of data from 8012 women in pregnant.371 women were SCH,7641 euthyroid. The results of this study was increased risk of GH,PROM and infants had increased risk of IUGR, LBW. Thus routine maternal thyroid functioning testing is needed to improve the maternal and fetal outcomes.

INDIAN

In 2013 Dhanwal D K et al reported prevalence of sub clinical hypothyroidism during 1st trimester was high -135/143 (North India 14.3%).

In 2006 Rao et al studied 163 non pregnant women with recurrent pregnancy loss in gestational age 12 wks in Hyderabad. This study was detected 4.2% prevalence of subclinical hypothyroidism. *In 2007 two government obstetrics and gynecology hospital in Chennai conducted study in prevalence of hypothyroid in pregnancy –the study reported subclinical hypothyroidism was 2.8% .*

In 2010 Sahu et al reported that prevalence of subclinical hypothyroid in pregnancy was 6.47% by this prospective analysis of 2nd trimester TSH level in 633 pregnant women. If TSH level was sub normal then free T4 and TPO Ab level were done. Patients were carefully monitored till delivery. Their obstetrical and perinatal outcomes were analysed. This study concluded increased adverse maternal and fetal outcomes. (cesarean section rate for fetal distress was high)

Vaidya et al 5 yr followup for 523pregnant women with no known thyroid disorder. TFT was done during antenatal period. In this study reported 12.4% of women was subclinical hypothyroidism in pregnancy.

In 2015 Rao S et al this study was conducted in ESI hospital, Hyderabad. 1062 pregnant women included and TFT done in routine antenatal clinic. He was reported 14.3% subclinical hypothyroidism and 6.2% overt hypothyroidism.

Nidhi Jalida Tinku et al 2014 this cross sectional study conducted in Bangalore. It includes 334 pregnant women of <14 wks gestation. This study was conducted in the urban population of southern India had adequate iodine status inspite this adequate iodine status prevalence of 9.2% subclinical hypothyroidism and 3.7% overt hypothyroidism.

SCREENING

Universal screening VS Targeted case finding

Vaidya et al reported that screening consider only women with high risk would miss 30%, so this study suggested that universal screening.

Dhanwal d k et al study in north india concludes that there is high prevalence of hypothyroidism 14.3% (135/143), majority being subclinical hypothyroidism, from this inferences, reported universal screening of hypothyroidism may be useful in our country.

Negro et al study not suggested universal screening- This study includes two groups – One is universal screening group consist of 484 women in high risk, 1798 women low risk, another group is targeted case finding group consist of 454 women in high risk and 1828 low risk.

This study did not show any statistically significant reduction in adverse outcomes with low risk women treatment with L Thyroxine in universal screening group.

According to ACOG SCH prevalence was 2 - 5%. This guideline states that benefit of treatment to either mother and fetus has not yet been demonstrated & routine screening for subclinical hypothyroidism in pregnant women is not currently recommended.

ADVERSE MATERNAL AND FETAL OUTCOMES:

In 1998, Leung AS et al study conducted in Los Angeles. here, 68 hypothyroid patients with no other medical disorders were analysed. 23 women with OH and 45 Women with SCH They were identified the adverse maternal outcomes of pre-eclampsia 22.5% OH/7.6%, gestational hypertension 36 % OH /25 %SCH. No adverse fetal and neonatal outcomes.

Wilson KL, Casey BM et al from November 2000 through April 2003- 24883 pregnant women were analysed for pregnancy hypertension. Results showed that 10.9% pregnancy hypertension was subclinical hypothyroidism group. This analysis concluded women with subclinical hypothyroidism identified during pregnancy have an increased risk for severe preeclampsia when compared with euthyroid.

Vander zanden et al showed that subclinical hypothyroidism women prone to get vascular complication of about 19.6% (hypertension during pregnancy) In the best study to date, Negro and colleagues suggested SCH increases the risk Maternal and fetal side effects.

Rajat Mohanty et al 2014 study concluded prevalence of subclinical hypothyroidism in south asia especially in India is more than other parts of world and mostly due to autoimmune and nutrition deficiency. The complications like abortion, preterm birth ,poor birth weight ,hypertension of pregnancy and convert to overt hypothyroidism.

Tudela CM et al conducted in study from November 2000 to April 2003 to estimate relation ship between sub clinal hypothyroidism and gestational diabetes about 24883 women.

Results showed that 23771 were euthyroid and 528 [2%] subclinical hypothyroidism. There was increased risk of GDM from 1.9% to 4.9% as thyrotropin increased from 0.001 to 10 milliunits/l. This study supports relation between subclinical hypothyroid and diabetes during pregnancy.

Jacmshid vafaeimanesh et al in 2011-2012 screened about 210 pregnant women (105 -with and 105 without GDM) suggested screening and treatment of subclinical hypothyroid in GDM. In GDM had 17.1% SCH/7 EH and in non GDM 7SCH/4EH.

Liang miao chen colleagues conducted study in china 2014, 8012 pregnant women included out this 371 was SCH, 7641 was euthyroid. Risk associated with SCH were GHTN-3.5SCH/1.8% EH, PROM 4.9% SCH/8.6% EH, IUGR 1.008% SCH /2.9 % EH and LBW 1.8% SCH/4.5% EH.

J clin endocrinol metab oct 2011 concluded that subclinical hypothyroidism was associated with increase risk of fetal distress, preterm labour and neurodevelopmental delay.

Abalovich et al showed that untreated hypothyroidism, subclinical hypothyroidism, clinical hypothyroidism at the time of conception is associated with miscarriage rate of 31.4% compared with 4% in euthyroid at conception of pregnancy complication like preterm labour, RDS, NICU care.

TPO Ab

Taka Mastu et al study conducted in 437 patients, found both types of auto antibodies TPO Ab and Tg positive in 316, only one in 85 and none in 36. Among patients positive for autoantibodies 50-70% were euthyroid, 25- 50% have subclinical and 5-10% clinical hypothyroidism.

In 2010 the study was conducted by De vivo A et al 216 pregnant women were enrolled. 176 women were euthyroid, 24 pts were found to have positive auto antibodies, 8 women were sub clinical hypothyroidism. women with subclinical hypothyroidism had significantly more risk for early pregnancy loss (p=0.02)

TREATMENT

Should subclinical hypothyroidism be treated in pregnancy.
ACOG 2010 –No ATA 2011 - yes Endocrine society 2012 - yes
Endocrine society suggested L Thyroxine treatment for all pregnant

women with subclinical hypothyroidism irrespective of TPO Ab. American thyroid association also suggested L thyroxine for pregnant women with subclinical hypothyroidism and positive TPO Ab.

European endocrinologist also suggested L thyroxine for subclinical hypothyroidism.

Beverly m shield et al 2013 conducted study in U K 523 women. In this study L thyroxine was given in all pregnant women with subclinical hypothyroidism and most case of subclinical hypothyroidism was resolved in postdelivery.so no need to continue L thyroxine post delivery.

PHYSIOLOGY OF THYROID

Iodine metabolism and increased Iodine requirement in pregnancy:

The average daily iodine requirement is 0.1mg.

Sources of iodine

Fish, milk, eggs and iodised salt are rich sources of iodine.

Absorption of iodine.

In the stomach and jejunum iodine is rapidly converted to iodide and absorbed into the blood stream.

Metabolism of Iodine:

Iodide actively enters the thyroid follicular cells by an ATP dependent process. Thyroid stores nearly 90% of body's iodine. The excess plasma iodine is excreted through the kidneys.

Thyroid hormone synthesis, secretion and transport:

The thyroid gland secretes two main hormones, Thyroxine T₄ and Tri iodothyronine T₃. T₄ is produced in greater quantities than T₃ (at a rate of 10:1) but T₃ is the major biologically active thyroid hormone and is mostly derived from T₄ in the peripheral tissue.

Iodine is obtained from the diet, converted to iodide, actively transported into the thyroid.

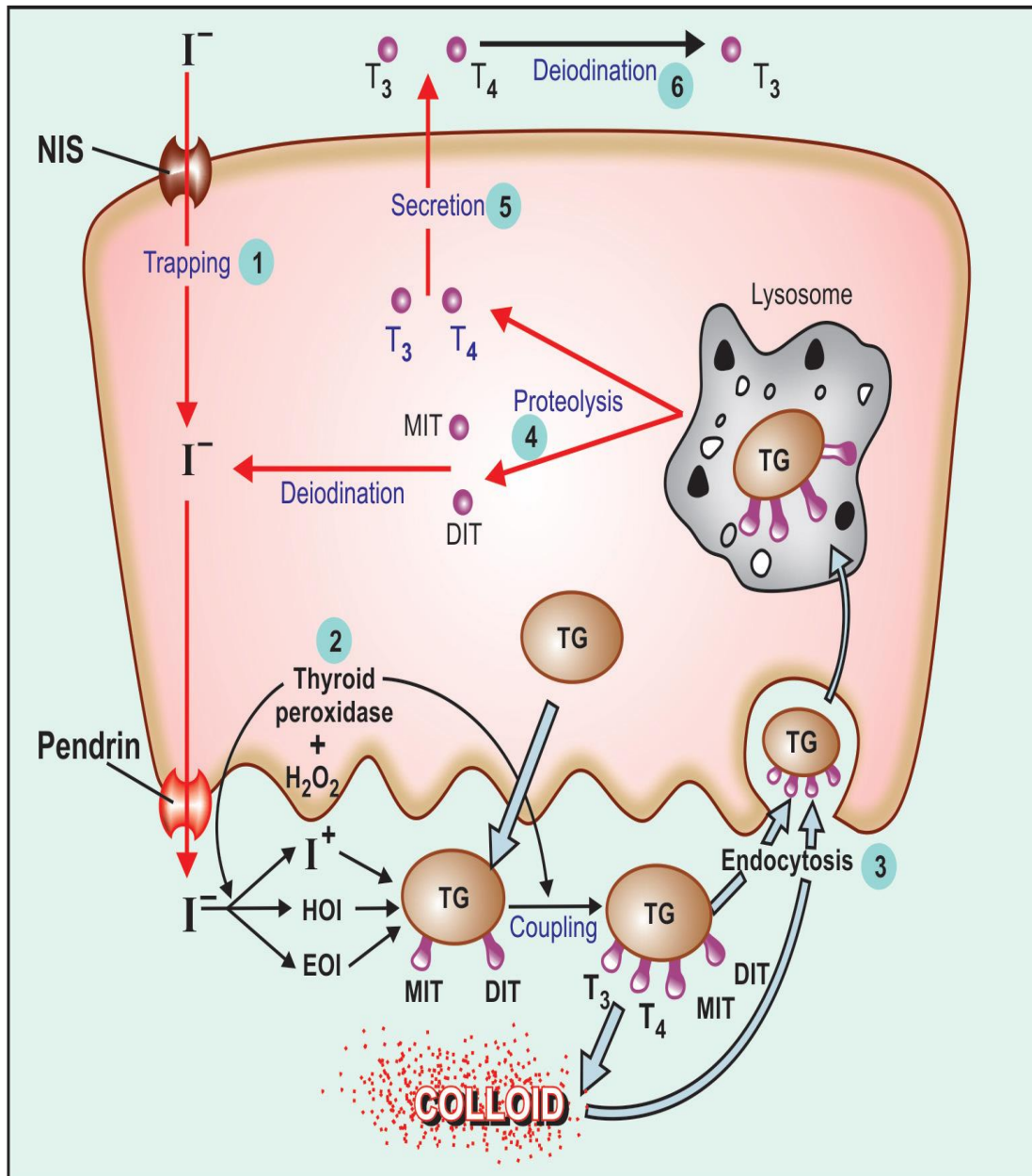
There are 5 steps in the production of thyroid hormones

- 1) Iodide trapping- iodide incorporated into the thyroglobulin by the way of the enzyme TPO. It is an ATP dependent active transport across the basement membrane of the thyroid follicular cells. The

thyroid follicles contain thyroglobulin (Tg) which is a glycoprotein with four tyrosyl residues.

- 2) OXIDATION -iodide oxidation to iodine followed by iodination of tyrosyl residues on the thyroglobulin. Both the step processes are catalysed by thyroid peroxidase. The end products of the second step are mono and diiodotyrosine. (MIT&DIT).
- 3) Coupling to form tetraiodothyronine (T4) or one moniodotyrosine and one diiodotyrosine molecule to form triiodothyronine (T3) or reverse triiodothyronine (rT3).
- 4) Hydrolysis of the thyroglobulin molecule to release free iodothyronines (T3andT4) and mono and diiodotyrosines.
- 5) Deiodination it yielding iodide which is reused by the thyrocyte.

MECHANISM OF THYROID SYNTHESIS



TRANSPORT AND STORAGE

Thyroid hormones are avidly bound to plasma proteins- only 0.03-0.08% of T4 and 0.2-0.5% of T3 are in the freeform. Thyroid hormone contain most of the protein bound iodine. T4 contain 90-95% PBI. The normal concentration of PBI is 4-10ug/dl. only 0.1-0.2% of this in T3, rest in T4.

Thyroid hormones are transported in serum bound form. T4 binds to 3 plasma protein in the following decreasing order of avidity.

1. Thyroxine binding globulin (TBG),
2. thyroxine binding prealbumin (TPBA -transthyretin) and
3. albumin.

Free form of thyroid hormone was small and is physiologically active hormone. T3 is three to four times more potent than T4. T3 is lower level in circulation than T4 and is less tightly bound to proteins. Hence it enters tissues more easily. T3 has a half life of one day while T4 has a half life of seven days.

Thyroid gland release thyroid hormone in the form of T₄ is 80% and in the form of T₃ 20% in euthyroid state.

H-P-T AXIS

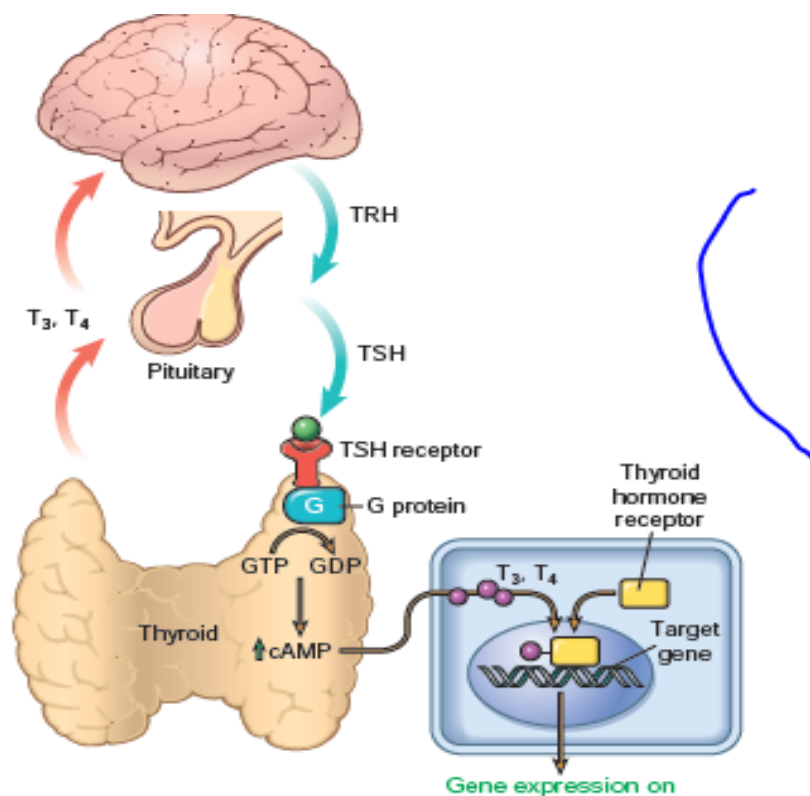


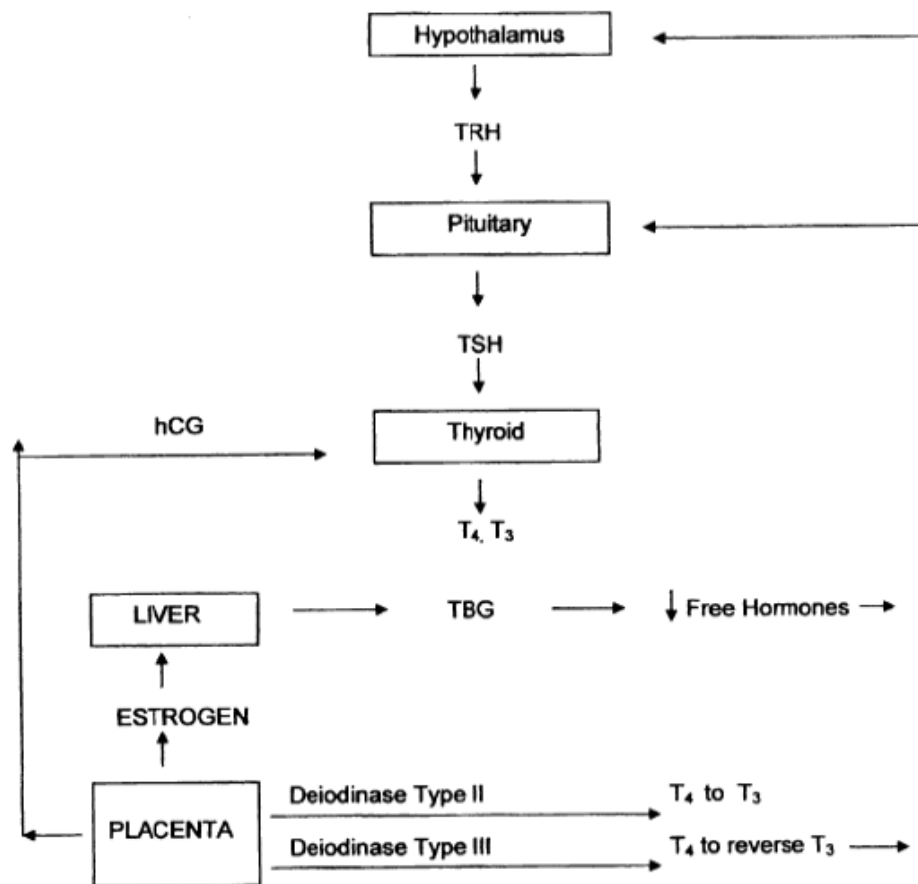
Figure 24-8 Homeostasis in the hypothalamus-pituitary-thyroid axis and mechanism of action of thyroid hormones. Secretion of thyroid hormones (T₃ and T₄) is controlled by trophic factors secreted by both the hypothalamus and the anterior pituitary. Decreased levels of T₃ and T₄ stimulate the release of thyrotropin-releasing hormone (TRH) from the hypothalamus and thyroid-stimulating hormone (TSH) from the anterior pituitary, causing T₃ and T₄ levels to rise. Elevated T₃ and T₄ levels, in turn, feed back to suppress the secretion of both TRH and TSH. TSH binds to the TSH receptor on the thyroid follicular epithelium, which causes activation of G proteins, and cAMP-mediated synthesis and release of thyroid hormones (T₃ and T₄). In the periphery, T₃ and T₄ interact with the thyroid hormone receptor (TR) to form a hormone-receptor complex that translocates to the nucleus and binds to so-called thyroid response elements (TREs) on target genes to initiate transcription.

H-P-T axis

Thyroid hormone production controlled by anterior pituitary by TSH-thyrotrophin.

TSH comprises two subunits and first one alpha subunit in common with LH, FSH and hCG and one specific beta subunit. TSH shows circadian and pulsatory secretion its secretion peaks at around midnight and declines during the day. The function of the pituitary is controlled by hypothalamus which secretes TRH .It accelerates the production of TSH whereas dopamine and somatostatin hinder it.

The thyroid hormones have a negative feedback effect on the pituitary and hypothalamus which is modified by the concentration in the serum and conversion of T4 to T3 locally in brain. Therefore, if T4 concentration in the serum drops, the inhibitory stimulus is decreased due to a diminished local effect of T3 in the pituitary and TSH levels rise to stimulate the thyroid gland (Ganong 2005, Hadley & Levine 2007)



Metabolism and excretion of thyroid hormone:

T₄ and T₃ metabolic inactivation occurs by glucuronide/sulfate conjugation and deiodination in Liver is the primary site, other sites are salivary glands and kidneys. The conjugates are excreted in bile. A major fraction is deconjugated in the intestines, reabsorbed by entero hepatic circulation and excreted in urine.

Mechanism of action:

Two form of thyroid hormone –T₄ &T₃ penetrates the cells by active transport and combines with a nuclear receptor .TR is bound to TRE in the enhancer region of target gene along with corepressors this leads to suppression of gene transcription. When T₃ binds to TR ,conformation changes occurred in TR and releasing the corepressors and binding the coactivator, then it induces gene transcription leads to production of specific m RNA and a specific protein.

Tachycardia, hypertension, arrhythmias, tremor, hyperglycaemia are clinical manifestations of thyroid hormone and are mediated partly if not completely by sensitisation of adrenergic receptors to catecholamines.

Functions of thyroid hormones:

By the process diffusion thyroid hormones enter the cells and binding of thyroid hormones to the nuclear receptors of cells results in translation and transcription of hormone specific genes. They affect almost every system in the body.

TH play a major role in fetal brain and skeletal development.

Growth and development

We understood TH action in growth and development by its action on the metamorphosis of tadpole to frog. Its action cannot be labelled catabolic or anabolic.

It exerts a critical control over protein synthesis. Deficiency of thyroid hormone mainly affects the nervous system in early life. In cretinism there is retardation and nervous deficit as a result of paucity of synapse formation axonal and dendritic ramification and reduced myelination. In pregnant with subclinical hypothyroidism also impairment of intelligence and slowing of movements in the offspring. (limited evidence)

Intermediary metabolism:

Thyroid hormones have an important role in carbohydrate, lipid and protein metabolism. It acts on cell membrane. T₄ has role in intermediary action.

Carbohydrate

It increases BMR

It increases the utilisation of carbohydrates due to the effect of increase BMR, gluconeogenesis and glycogenolysis compensate for it. So hyperglycaemia and a diabetic like state occur in hyperthyroidism.

Protein:

T4 has catabolic role overall. Negative nitrogen balance and tissue wasting result from prolonged action, it leads to weight loss in hyperthyroidism and weight gain in hypothyroidism. Thyroid hormones inhibit mucoprotein synthesis in low concentrations which characteristically accumulates in myxedema.

Lipid:

T4 and T3 indirectly enhance lipolysis by suppressing a phosphodiesterase although lipogenesis is also stimulated.

Cholesterol metabolism is accelerated but its conversion to bile acids dominate. Thus hypocholesterolemia is a feature of hyperthyroidism and hypercholesterolemia and obesity are features of hypothyroidism.

Calorigenesis:

Basal metabolic rate is increased by stimulation of cellular metabolism and resetting of the energy stat. But the metabolic rate in uterus, gonads, brain, spleen and lymph nodes is not significantly affected. The mechanism is uncoupling of oxidative phosphorylation thus releasing excess energy as heat.

Cardio vascular system

T4 and T3 causes hyperdynamic state.

Heart rate, contractility and output are increased which cause a fast and bounding pulse. Thyroid hormones act on the contractile elements of the heart and stimulate them by upregulation of beta adrenergic receptors. They have a positive inotropic and chronotropic effect. Actions of catecholamines are augmented. That is why fibrillation and arrhythmias are common in hyperthyroidism. It can also precipitate angina. Blood pressure particularly systolic is often raised. Myocardial oxygen consumption can be markedly reduced by induction of hypothyroidism.

Nervous system:

It act on respiratory centre of brain and maintain the normal hypoxic and hypercapnic drive .Mental retardation is the hallmark of cretinism. Sluggishness and other behavioural features are seen in myxedema whereas tremors, anxiety and hyperreflexia are seen in hyperthyroidism.

Skeletal muscle:

Thyroid hormones increase the protein turnover and speed of muscle contraction and relaxation. Myxedema is characterised by flabby

and weak muscles while thyrotoxicosis causes an increase in muscle tone, tremor and weakness due to myopathy.

Gastro intestinal system:

Thyroid hormones increase gastric motility. Hypothyroid patients are often constipated while diarrhea occurs in hyperthyroidism.

Kidneys

They have no effect in euthyroid individuals but cause diuresis when myxedematous patients are treated with them.

Haemopoiesis:

Anaemia occurs in hypothyroid individuals, thus it is proven that T4 plays a role in erythropoiesis.

Reproduction:

Oligomenorrhoea and subfertility/infertility is known to occur in women with hypothyroidism. Normal functioning of the thyroid gland is essential for maintenance of pregnancy and lactation.

Thyroid changes in Pregnancy:

Anatomically, the thyroid gland undergo moderate enlargement which is caused by glandular hyperplasia and increased vascularity during pregnancy. Mean thyroid volume increased from 12 ml in first trimester to 15 ml at delivery. Total volume of is inversely proportional to serum thyrotropin concentration, (so any goiter should be investigated) normal pregnancy does not cause significant thyromegaly.

Several alteration in thyroid physiology and function during pregnancy. In 1st trimester–TBG increase, reach their zenith at about 20wks and stabilize at double baseline value for the remainder of the pregnancy.

Increased TBG–higher hepatic synthesis due to estrogen (linderberg et al 1974) and lower metabolism rate of TBG sialylation and glycosylation and reduced renal clearance(Ain et al 1987) Effect of increased TBG due to increased binding of T4 to TBG (Robbins & Nelson 1958) so Total serum T3 and T4are increased. Biologically active free T4are slightly increased and peak along with hCG. Total T4 sharply increasing between 6 and 9 wks and reaches plateau at 18 wks. (Glinioer et al 1990)

Thyroid hormone is derived from iodination of tyrosine residues in thyroglobulin to form mono or di iodo tyrosine which are then coupled to form T4 and T3.

Role of deiodinase:

Free T4 is metabolized in the tissues to the active form free T3 by three deiodinase enzymes. The tissues have different rates of free T3 production and uptake according to the presence of the deiodinase enzymes.

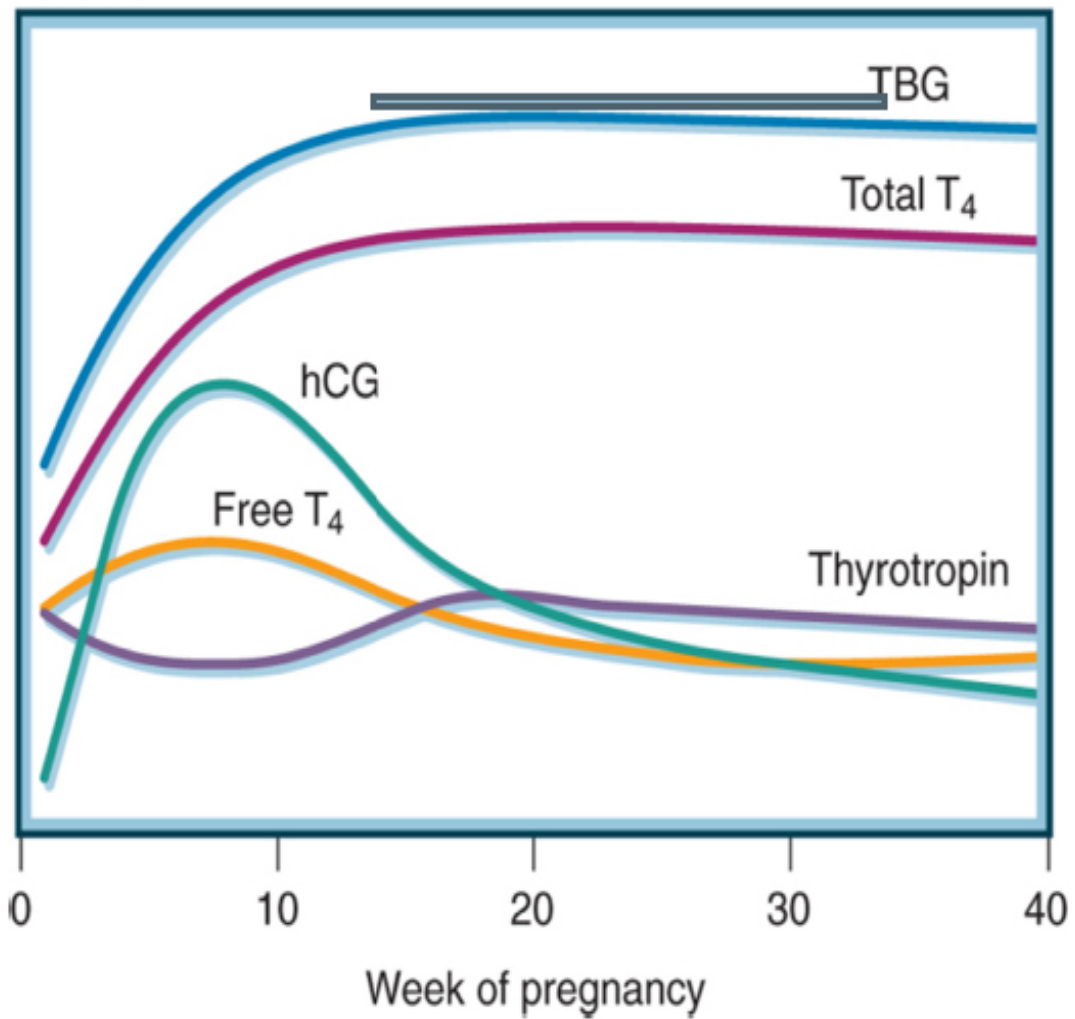
Type 1 – liver, kidney, thyroid and pituitary- $fT3$ formation-activity low in fetus.

Type 2- CNS and pituitary-supply $fT3$ to the brain

Type3- Brain and reproductive tissues-inactivates both $fT3$ and $fT4$.

Only D2 and D3 have been detected in placental tissue.D2-providing the placenta with supply of $fT3$ and D3 maintaining equilibrium.

Mother



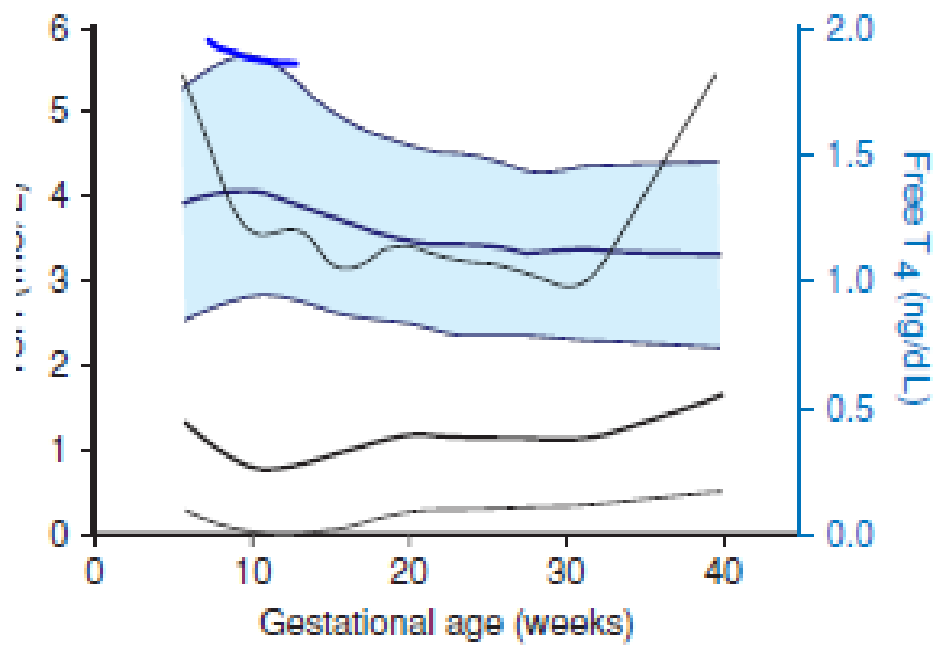
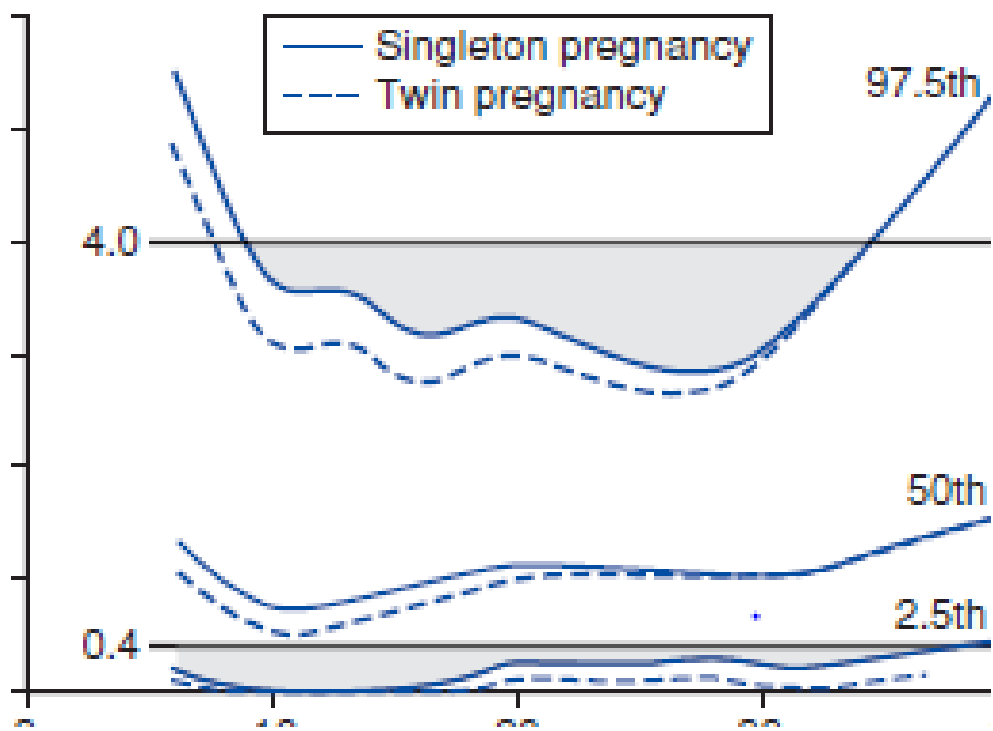
During normal pregnancy, the maternal thyroid hormone production increased more than 50%.

First-Following conception, estrogen concentrations increase markedly (lindberg et al 1974) leading to increased production of TBG.

The increased binding of thyroid hormone together with its increased metabolism by the placenta, developing fetus utilizes the maternal TH, increased distribution volume of T4 leads to a greater requirement for thyroid hormone production in order to maintain free T4 levels. Total T4 levels are above the normal non pregnant levels. Women who have borderline iodine deficiency may be unable to meet this increased demand resulting in reduction in thyroid hormone product. So HCG has weak TSH like activity.

In early pregnancy Serum thyrotropin (TSH) level is decreased because of thyroid stimulation from the weak TSH effects of HCG. So there is also a slight increase in free T4 levels. Molar gestation, hyperemesis gravidarum or multiple gestation are conditions associated with higher than usual HCG levels. These may result in an exaggerated stimulation of the thyroid gland and produce transient first-trimester thyrotoxicosis. (glinioer 1997) TSH will rebound to normal nonpregnant levels once HCG returns to a steady state.

So there will be a mild decline in free T4 and an increase in TSH after the first trimester. But these changes typically remain within the reference range. That is why FT4 and TSH levels should be interpreted after comparison with specific reference ranges for each trimester



TSH LEVEL IN PREGNANCY

Cut off values for TSH in pregnancy:

Non pregnant TSH reference range are unreliable in pregnancy due to physiological changes of thyroid.- suppressive effect of increasing thyroxin and increased TSH excretion, TSH is kept at its lowest minimal level or can even go below normal range.

Trimester specific values as per ATA2011,ES2012 and USPSTF GUIDELINES.

TRIMESTER	TSH(miu/dl)	fT4(ng/dl)
1 trimester	0.1-2.5	0.8-1.2
2 trimester	0.2-3	0.6-1.0
3 trimester	0.3-3	0.5-0.8

Glioner et al –S TSH >2miu/ml and TPO Ab >1250u/ml before 20 weeks predict the occurrence of hypothyroid in later trimester of pregnancy

Thyroid physiology in the fetus and neonate:

Development of brain, lung and bone in the fetus is absolutely dependent on maternal thyroid hormone.

The human fetal thyroid metabolically inactive until 9 and 12 wks, starts to concentrate iodine at 11 wks and is able to produce TG is precursor of TH at 4 week.

At 18 wks the fetal HPT axis fully developed, mature follicles in thyroid gland was developed and it can start to produce TH of its own. (Burrow et al 1994) * TSH levels of the fetus continue to increase till 28 weeks after that it reaches a nadir and remains at the level till term.

Free T₄ concentration increases progressively till term and exceed maternal levels. Hence, some level of fetal hyperthyroidism exists at term.

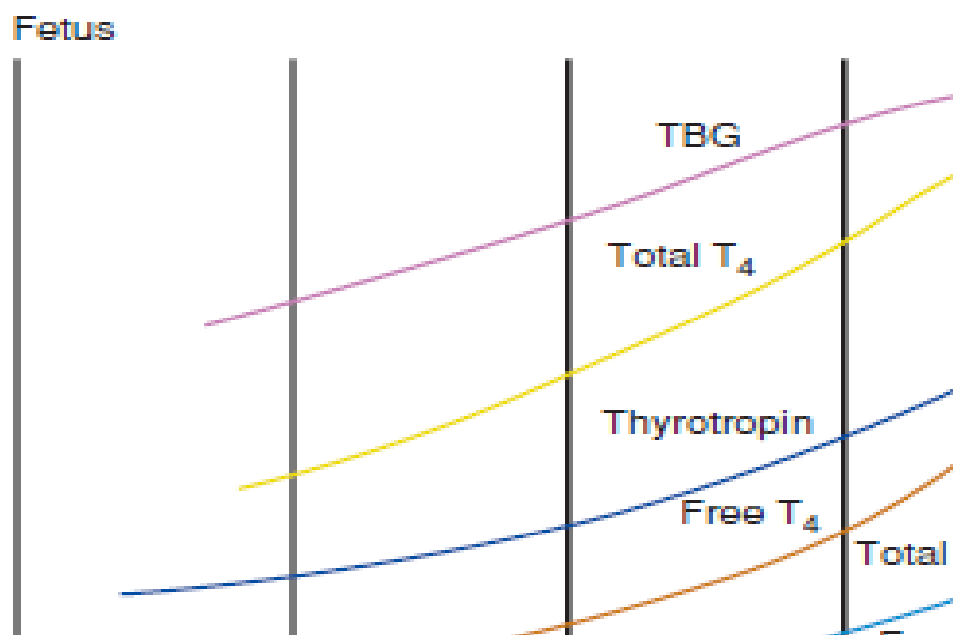
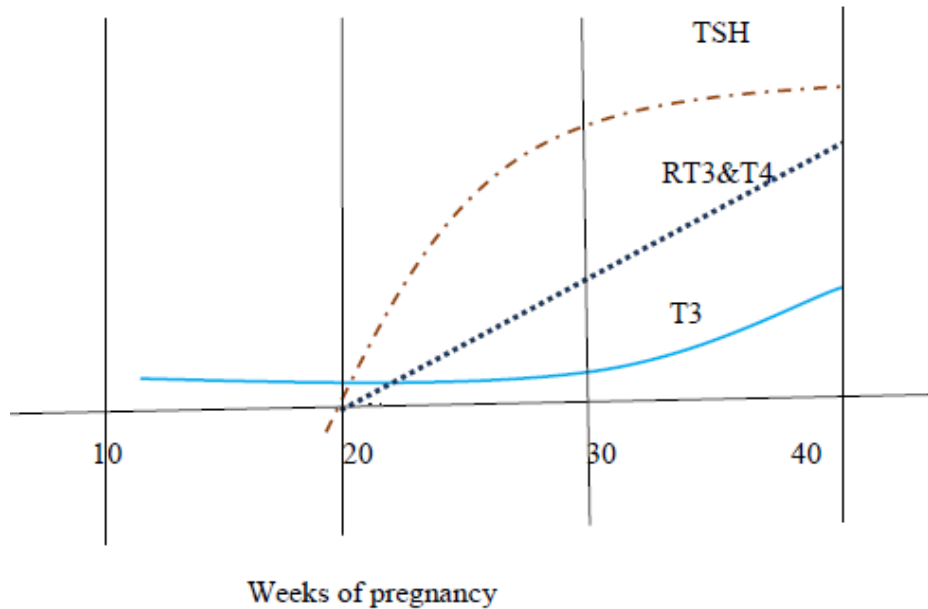


Fig: FETAL THYROID PHYSIOLOGY



Iodine from T3 & T4 removed by D3placental deiodinase and generating inactive iodothyronines and reverse T3. Large amounts of T4 crossing the placenta prevented by it. Another reason for increase in iodine demand is increased GFR with an increased urinary clearance of iodine. Although the fetal thyroid starts developing by 12 wks of gestation, it cannot organify iodine till 20wks of gestation. Till that period the maternal T4 is the only form of the hormone that can cross the placenta. Deiodinase enzyme in the fetus converts maternal fT4 to T3 in brain & other tissues. So the fetal iodine store solely demands on maternal intake during this period.

In 1st trimester total T4 concentration in fetus is directly related to mother T4 concentration T3 level lower than rT3.

rT3 level higher because of D3 in the placenta, which acts as barrier and protects the fetus from TH excess(calvo et al 2002) Fetus has high free TH levels due to the lower level of binding protein.

D2 and D3 activity which ensures them a supply of fT3 .(burrow et al. calvo et al, 1994, 2002)

T4 is the major fetal thyroid hormones.

In labour the fetus goes from a state of relative T3 deficiency to T3thyrotoxicosis.

Immediately after birth the TSH rise rapidly & falls to basal levels after 48-72hrs of birth. Because of that T4 and T3 levels increase and reach the peak values by 24-48 hours and 24 hours of age respectively. Hyperactivity of thyroid disappear at 3-4 wks above changes occur mainly due to TRH surge as a response to rapid neonatal cooling since the TSH surge is accompanied by a prolactin surge. These thyroid changes are believed to be a protective mechanism against sudden entry of cool environment.

Subclinical Hypothyroidism:

By definition, it is a condition in which TSH is elevated ,but FT4 is normal.

Incidence of subclinical hypothyroidism is at least 2.5% .Usually it is asymptomatic, but there is evidence autoimmune thyroid disease (positive TPOAbs and or TG antibodies) in 50-60%(12) Subclinical hypothyroidism was found to be more common in women delivering before 32 weeks.(12)

Pregnancies complicated by subclinical hypothyroidism had a 3 fold increased risk of developing placental abruption and 2 fold increased risk of preterm labour compared to euthyroid women.(22)Gestational hypertension also occurred more commonly in these women.(23)

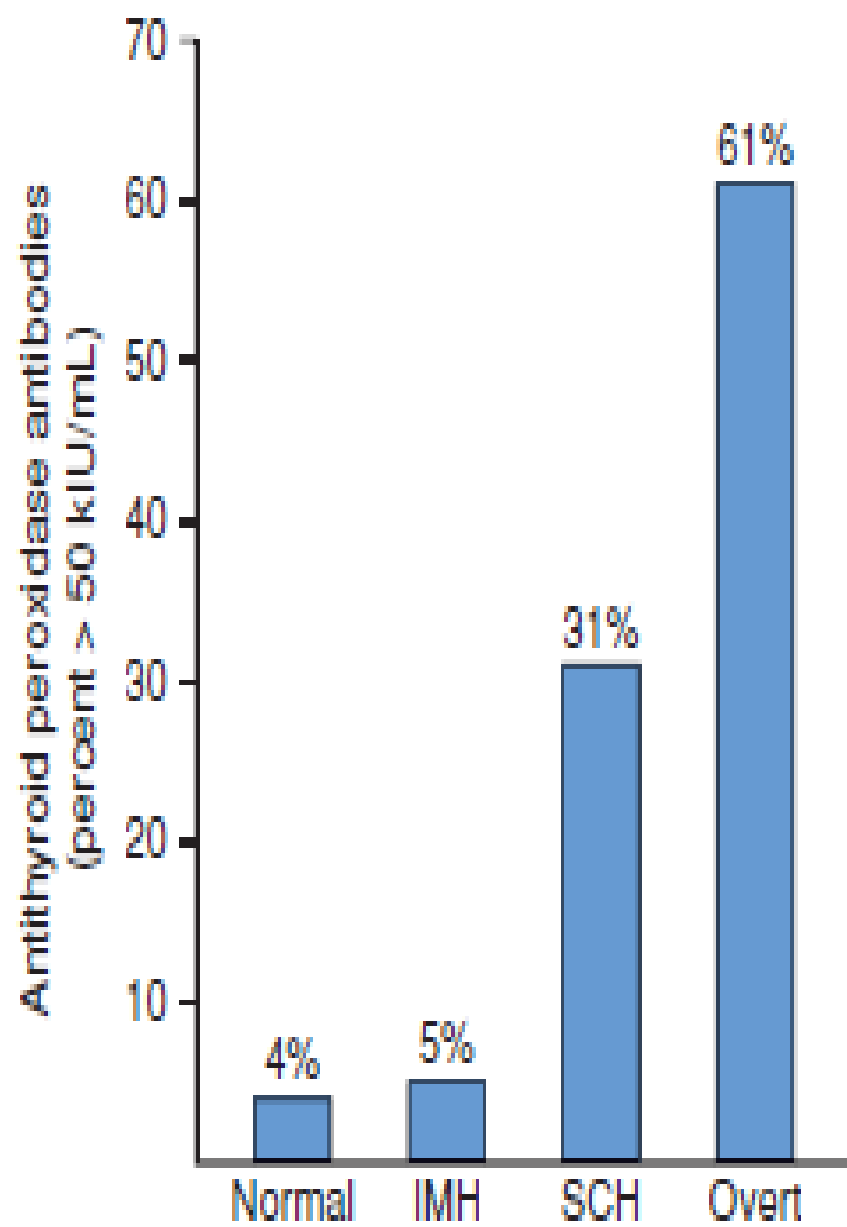
Even raised maternal TSH (high level of normal) is associated with neonatal respiratory distress, miscarriage and preterm delivery(12).The likelihood of patients diagnosed as hypothyroids during pregnancy to continue to be hypothyroid even after pregnancy depends on the initial TSH value. The United States Preventive Services Task force reported that nearly almost all patients with an initial TSH >5mIU/ml developed overt hypothyroidism within 5 years.(33)

TABLE 58-5. Pregnancy Outcomes in Women with Untreated Subclinical Hypothyroidism and Isolated Maternal Hypothyroxinemia Compared with Euthyroid Pregnant Women

Outcome	Euthyroid n = 16,011	Subclinical Hypothyroidism n = 598	p value	Isolated Hypothyroxinemia n = 233	p value
Hypertension (%)	9	9	0.68	11	0.53
Placental abruption (%)	0.3	1.0	0.03	0.4	0.75
Gestational age delivered (%)					
≤ 36 weeks	6.0	7.0	0.09	6.0	0.84
≤ 34 weeks	2.5	4.3	0.005	2.0	0.44
≤ 32 weeks	1.0	2.2	0.13	1.0	0.47
RDS/ventilator (%)	1.5	2.5	0.05	1.3	0.78
Neonatal intensive care (%)	2.2	4.0	0.005	1.3	0.32

RDS = respiratory distress syndrome.

Data from Casey, 2007.



Treatment of subclinical Hypothyroidism:

According to USPSTF, AACE, COCHRANE REVIEW recommends treatment for pregnant women with SCH.

HISTORY

Thyroid hormone replacement was first described by Murray in 1891 by injecting *sheep* thyroid extract.

Purified thyroxine crystals were identified by Edward Calvin Kendall in 1914.

Structure of thyroxine was identified by British chemist Harrington in 1926.

Drug of choice is oral levothyroxine, as it is category A drug. Levothyroxine or T₄, normally secreted by the thyroid follicular cell.

Levothyroxine is the synthetic form of thyroxine

For therapeutic purposes L thyroxine is superior to lio thyronine because of its longer duration of action, it has a long half life 7 days, and is partially converted to T₃ in the body, resulting in constant physiological level of T₃ and T₄.

The only accepted indication for the use of liothyronine is myxedema coma where a quick response is essential.

USPSTF GUIDELINES

Levothyroxine should be taken on an empty stomach either 30 to 60 minutes before breakfast or at bedtime, with water at the same time each day.

Patients should avoid taking calcium and iron within 4 hrs of thyroxine. Increase in dosage is required during pregnancy.

2014 European thyroid association guidelines-ETA.

SCH arising during conception and preconception should be treated with levothyroxine.

Before conception pt on Levothyroxine, 25-50% increase in dosage is required during pregnancy.

Newly diagnosed SCH during pregnancy initiating the treatment with following dosage

TSH < 4.2 mIU/dL – Levothyroxine (T4) dose - 1.20 µg/kg/day

TSH > 4.2 to 10 mIU/dL - Levothyroxine (T4) dose - 1.42 µg/kg/day

It should be started as a low dose of 12.5ug-25ug, maintenance dose should be 2-2.4ug/kg/day.

TSH level should be monitored every 4-6 wks during first trimester, once in second and third trimester, titrate the dose rapidly to keep the TSH at a level less than 2.5 IU/ml in the first trimester and less than 3 IU/ml in second and third trimesters.

The association urges physicians to be vigilant in identifying and treating women with subclinical thyroid dysfunction before conception.

The Journal of Clinical Endocrinology and Metabolism published an Executive Summary which recommends thyroxine replacement in women with subclinical hypothyroidism.

The goal of LT₄ treatment is to normalize maternal serum TSH values within the trimester-specific pregnancy reference range (first trimester, 0.1–2.5 mIU/L; second trimester, 0.2–3.0 mIU/L; third trimester, 0.3–3.0 mIU/L). **Level A-USPSTF**

2014 ETA GUIDELINES

Role of Iodine in SCH

In pregnancy there is about a 50% increase in iodine requirement to achieve a dietary intake of 250 ug/day. This increase is due to an increased glomerular filtration and renal iodine clearance as well as iodine trans placental transfer to the fetus particularly in later gestation.

The contribution of iodine deficiency to the incidence of SCH is variable.

The Spanish study did show high prevalence of SCH in iodine deficient area in Belgium.

Iodine prophylaxis in SCH has not been studied.

Prevention of endemic goiter and presumably some case of SCH can be affected by iodine supplementation.

According to the WHO ,pregnant and lactating women should be provided with 250 ug /day iodine. this should be taken as in the form of potassium iodide.

Should not exceed 500 ug .

The effectiveness and side effects of iodine prophylaxis together with or without L thyroxine therapy in subclinically hypothyroid women should be assessed.

Complications of sub clinical hypothyroidism in pregnancy:

Maternal

- ☐ Spontaneous abortion
- ☐ Pregnancy induced hypertension(pre eclampsia, eclampsia)
- ☐ Placental abruption
- ☐ IUGR
- ☐ Oligohydramnios
- ☐ Preterm delivery
- ☐ Fetal distress
- ☐ Low birth weight

Congenital Hypothyroidism:

Congenital Hypothyroidism (CH) is one of the most common preventable causes of mental retardation. The incidence is 1:4000 livebirths and the worldwide and the incidence in India is 1:2500-2800 live births.

Thyroid dysgenesis is the commonest cause attributing for the majority of cases. CH can be permanent or transient. Maternal cytotoxic antibodies and genetic mutations causing inactivation of thyroid receptor can be a cause. There is clinical and scientific evidence that hypothyroxinemia causes poor neurodevelopment outcome in the children of mothers with low thyroxine levels.

In a study by Morreale de Escobar et al in 2004, it was noted that thyroid hormone accumulates in the cerebral cortex before 20 weeks.(30)

Primary evidence of the effect of the deficiency of thyroid hormone on cerebral cortex was studied by Lavado-Autric et al in 2003.(31)

Defects in thyroid hormone synthesis account for 10% of all cases. These can be inherited as autosomal recessive disorders. Pharoah et al did a landmark study in 1971 and came to a conclusion that iodine supplementation in pregnancy prevented subsequent cretinism.

Early and aggressive treatment with thyroxine is crucial for infants with congenital hypothyroidism. Yet some infants with prompt replacement exhibit mild cognitive defects in adolescence.(47)

CLINICAL FEATURES OF CONGENITAL HYPOTHYROIDISM

Untreated severe congenital hypothyroidism leads to irreversible growth failure and mental retardation

- ☐ Feeding problems, constipation, growth failure and hoarse cry are early symptom.
- ☐ Dry skin and decreased growth of nails and hair; delayed tooth eruption are delayed symptoms
- ☐ Closure of anterior and posterior fontanelles is delayed
- ☐ Cardiomegaly may be present

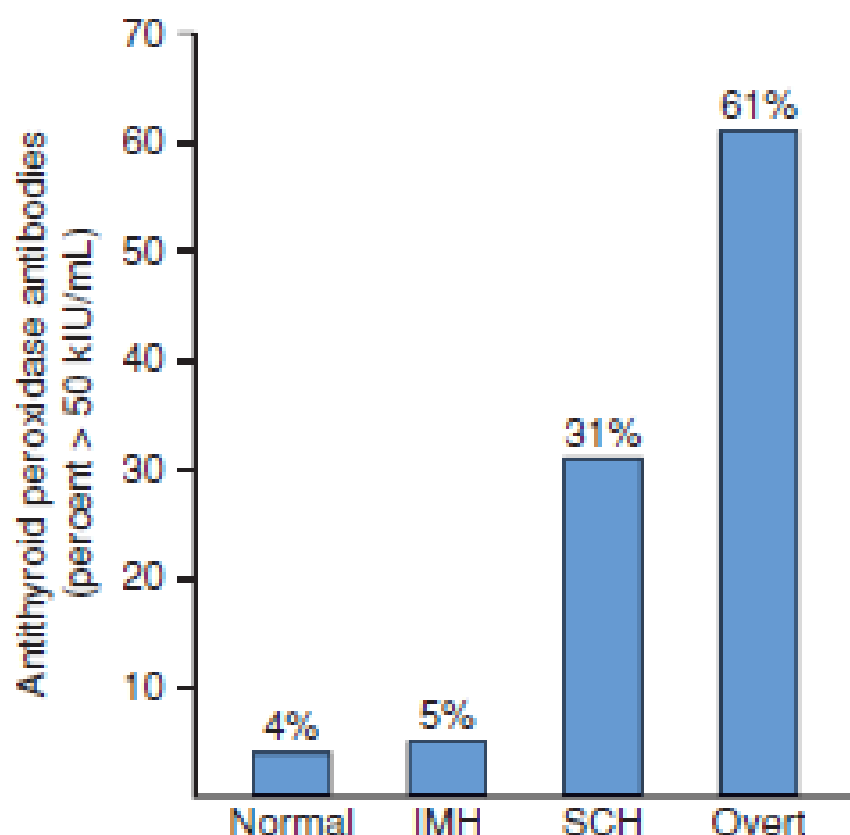
The other clinical features are broad, flat nose, pseudohypertelorism, puffy, myxedematous facies, large, protruding tongue, prolonged neonatal jaundice, protuberant abdomen, umbilical hernia.

Thyroid antibodies:

☐☐ These tests do not determine the thyroid function, instead they indicate the underlying disorder. Antithyroglobulin, antimicrosomal and thyroid stimulating immunoglobulin are the thyroid antibodies. Approximately 80% of patients with Hashimoto's thyroiditis have raised thyroid antibody levels.

- □ Thyroid peroxidase (TPO)antibodies and Anti Thyroglobulin (TG) antibodies are associate with pregnancy complications. There are studies to show that euthyroid women with recurrent miscarriages and preterm birth were found to have antibodies to either TPO or TG.TPO antibodies have also been implicated in the development of postpartum thyroiditis.
- □ Association between auto immune thyroiditis and adverse obstetric outcome independent of thyroid function has also been proven in another prospective study where euthyroid TPO antibody positive women who received thyroxine supplementation in early pregnancy had a reduced rate of miscarriage and preterm delivery rate(12)
- □ Pregnant women with TPO antibodies were found to have a three times more chances of placental abruption when compared with antibody negative controls in a study by Abbassi-Ghanavati et al in 2010.
- □ Pop et al revealed decrease in the intelligent quotient of children aged 5 years whose mothers were TPO antibody positive at 32 weeks of gestation even though they were actually euthyroid.(4)

- □ Some thyroid autoantibodies cross the placenta causing fetal thyroid dysfunction. But maternal Hashimoto thyroiditis is not typically found to be associated with fetal thyroid abnormalities.
- □ Brown and co-workers in 1996 did a study on over one million babies and found that only 1 in 180,000 neonates born to mothers with Hashimoto's thyroiditis had thyroid *dysfunction*. (46) Stagnaro Green and Glinow studies reported TPO Ab positive women had high incidence miscarriage and preterm labour.



Screening for thyroid dysfunction during pregnancy:

For universal screening to be recommended for a disease,

- ☐ ☐ The incidence of the disease should be high enough to warrant screening.
- ☐ ☐ The screening needs to be as cost effective as possible
- ☐ ☐ There should be substantiative evidence that adverse outcomes are associated with the disease.
- ☐ ☐ There should also be evidence that intervention improves outcomes.

The journal of clinical endocrinology and metabolism adopted a clinical practice guideline in 2007 which recommended screening among the following high risk women(27)

- a) Women with a previous history of hyper /hypo thyroid disease / thyroid ectomy/goitre
- b) Women with family history of thyroid dysfunction
- c) Women with symptoms/signs suggestive of thyroid dysfunction
- d) Women with autoimmune diseases like Type 1 DM

- e) Women with a history of infertility
- f) Women with history of head and neck irradiation
- g) Women with history of recurrent miscarriages or preterm deliveries.

According to the ACOG Committee Opinion no.381(oct 2007) also thyroid screening in pregnancy should be carried out only on symptomatic women/those with a history of thyroid disease or other medical illnesses that may be associated with thyroid disease (eg:diabetes)

In a study by Bijay Vaidya et al published in JCEM in 2007, they found that thyroid function testing of only high risk women would miss about 1/3rd of women with overt/subclinical hypothyroidism.(21)

AIMS OF THE STUDY

Primary Aim:

To assess the benefits and pregnancy outcome in promptly diagnosed and adequately treated antenatal hypothyroid women.

Secondary Aims:

To assess whether unfavourable pregnancy outcome and complications are more among the antenatal women who are diagnosed late in pregnancy and hence inadequately treated.

MATERIALS AND METHODS

To measure pregnancy outcomes in all the study subjects, all antenatal women in their first trimester (first booking) or if they have their first visit only in second and third trimester will be screened with serum TSH. If TSH is more than 3miu/ml, they will be started on treatment after doing an FT4. They will be monitored to see if their treatment is adequate by repeating a serum TSH again in 2nd and 3rd trimesters. These women will be followed up till term and monitored for any complications. Finally, they will be analysed to see if there is a significant increase in complications in the antenatal women who were diagnosed and started on treatment late and whether these complications could have been avoided if they were to be started on treatment early in the course of pregnancy.

Inclusion Criteria:

- a) All first and second trimester antenatal women with singleton gestation which will includes, family history of hypothyroid, auto immune disease. History of treated PCOD, RPL, miscarriage, presence of TPO Ab, Type 1 Diabetes.

Exclusion Criteria:

- Multiple gestation
- Overt hypothyroidism
- Thyroidectomy

USPSTF 2011

TSH is the more accurate indication of thyroid status in pregnancy than any other (f T4,thyroid Ab)

ESTIMATION OF TSH**Method**

Solid Phase Two-Site Immuno Radio Metric Assay (IRMA) with IRMAK-9 kit, BRIT, Mumbai.

Results

Normal range of TSH was taken as 0.17 – 3 μ U/ml. (Trimester Specific)

ESTIMATION OF FREE T4

Method

RIA (Radio Immuno Assay) – IMMUNOTECH

Results

Normal range of Free T4 was taken as 0.8 -1.7 ng/dl (Trimester Specific)

ALEXANDER recommends TPO Ab screening in SCH

ESTIMATION OF TPO Ab

Method

Chemi luminescent immune assay

Results

Positive >34IU/ML

Negative<34IU/ML

STATISTICAL METHOD

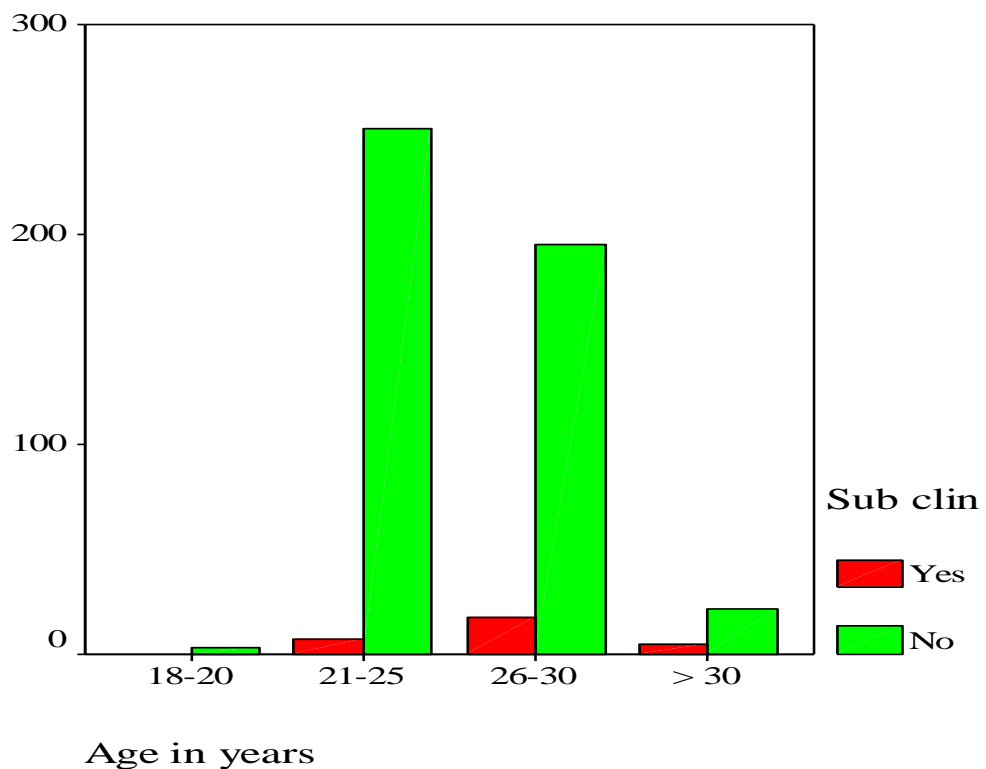
The statistical package which was used for doing the analysis was SPSS 16.00 version. Raw data were analysed using cross tabulations, Chi square test and p value.

OBSERVATION AND RESULTS

AGE DISTRIBUTION

Age in years	Number		Percentage	
	Eu TH	SCH	Eu TH	SCH
18-20yrs	3	0	0.6	
21-25 yrs	250	7	53.2	
26-30yrs	195	18	41.5	
>30yrs	22	5	4.7	

Higher incidence of SCH in 26-30 yrs of age

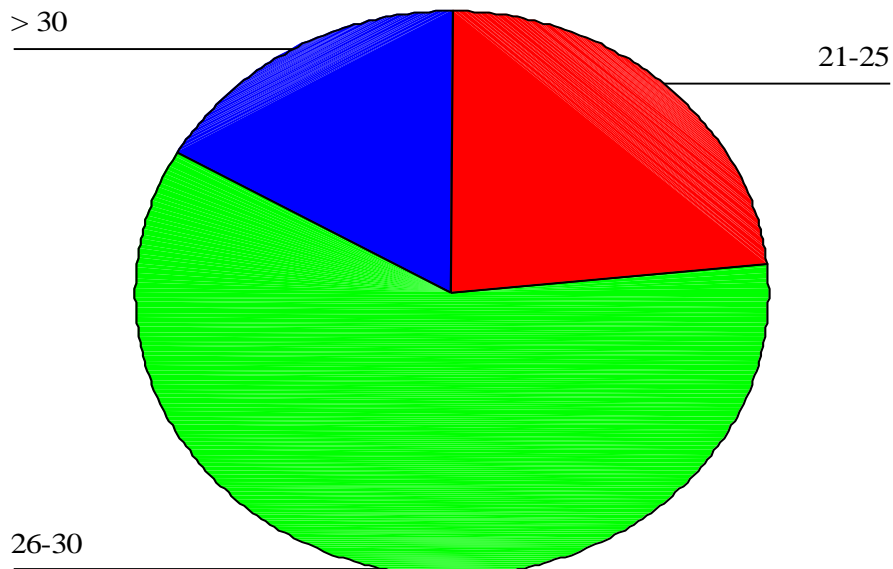


SCREENING AT TRIMESTER

TRIMESTER	NUMBER		PERCENTAGE	
	EuTH	SCH	EuTH	SCH
1	256	17	54.5%	56.7%
2	139	8	29.6%	26.7%
3	75	5	16%	16.6%

More than 55 %women screened at 1st trimester in my study.

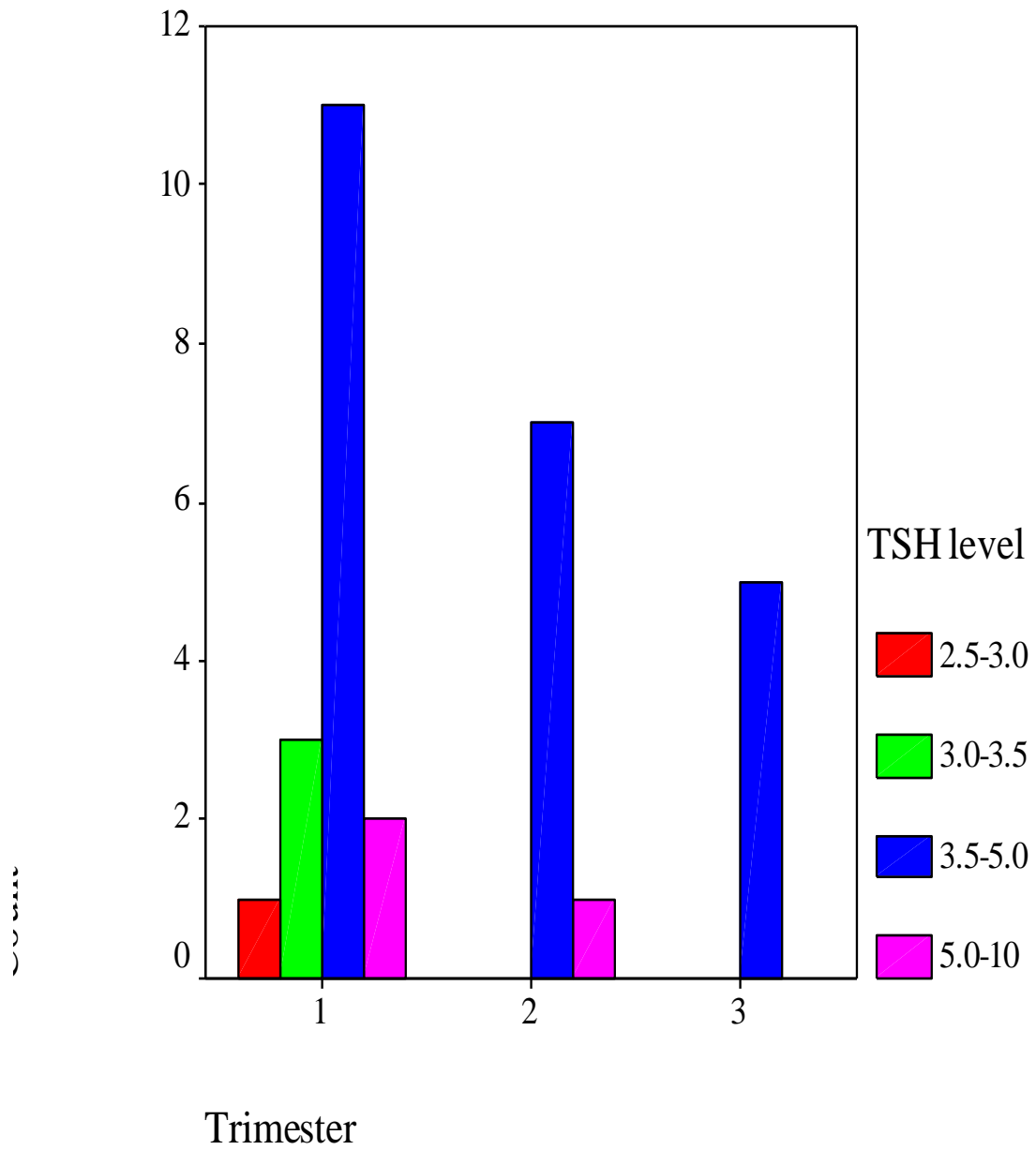
Age in years



TSH AT DIAGNOSIS OF SCH

TRIMESTER		TSH LEVEL		
	2.5 -3uiu/l	3-3.5	>3.5-5	>5 -10
	No %	No %	No %	No %
1	1 5.9%	3 17.6%	11 64%	2 11.8%
2	-	-	7 87.5%	1 12.5%
3	-	-	5 100%	-

Most of the SCH women TSH range 3.5 -5 miu/dl

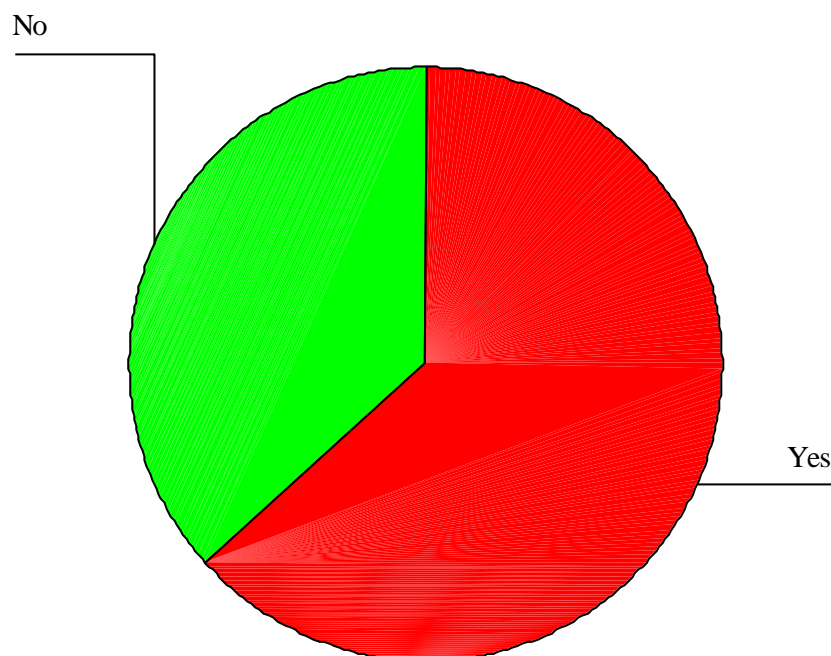


ADEQUATE AND INADEQUATE TREATMENT

TREATMENT	NUMBER	PERCENTAGE
Adequate	13	43.3%
Inadequate	17	56.7%
Total	30	100

13 women were adequately treated in my study.

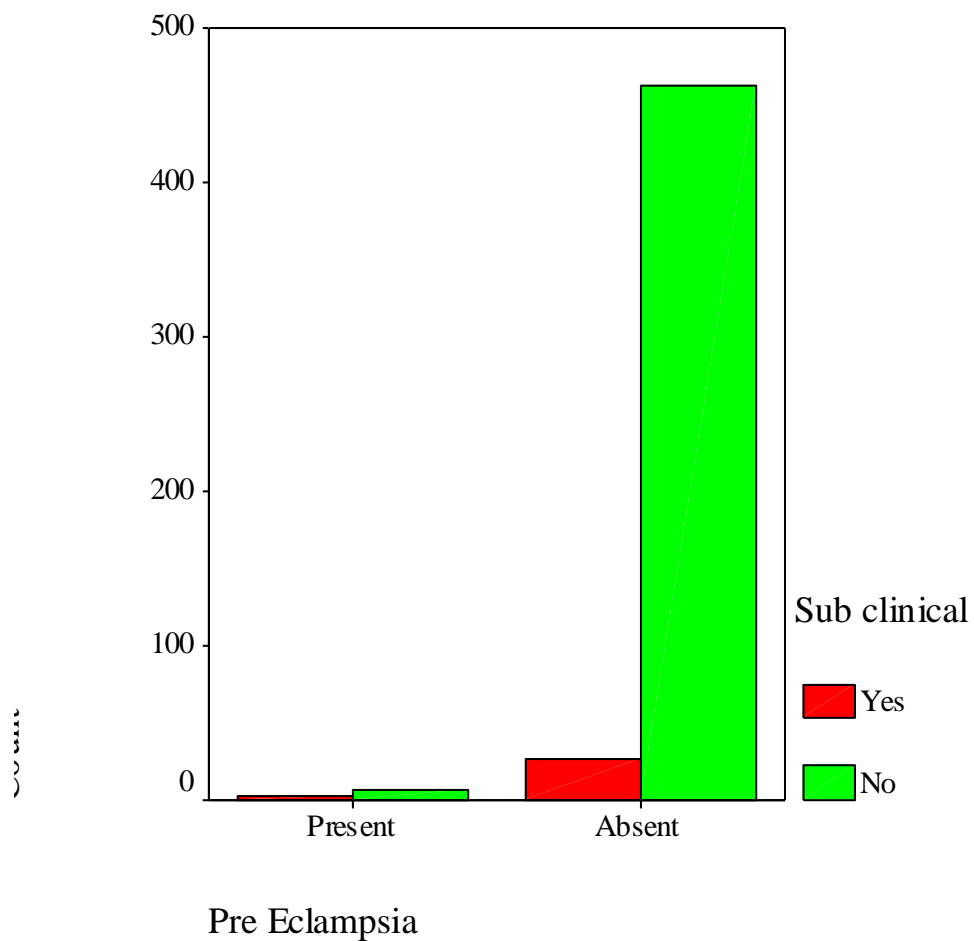
Treatment



PREVALENCE OF PREECLAMPSIA IN EuTH AND SCH

Pre Eclampsia	Number		Percentage	
	EuTH	SCH	E uTH	SCH
Present	16	3	3.4 %	9%
Absent	454	27	96.6%	91%
	470	30	100	100

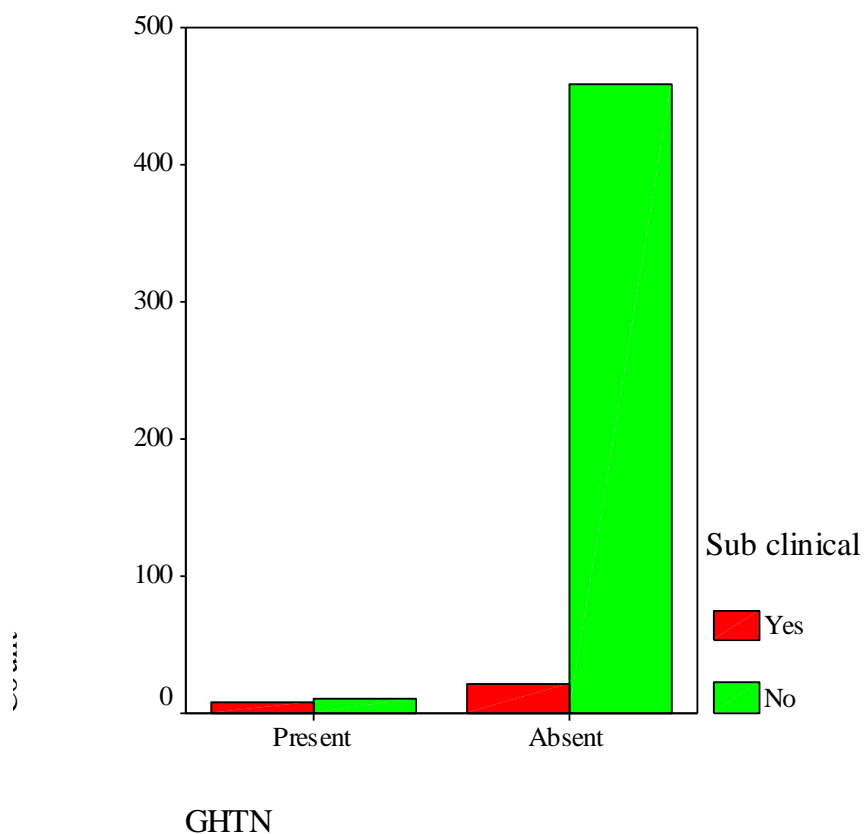
The incidence of preeclampsia in euthyroid women was 3.4% and 9% in SCH women.



PREVALENCE OF GHTN IN EuTH AND SCH

GHTN	NUMBER		PERCENTAGE	
	EuTH	SCH	EuTH	SCH
Present	23	3	4.8%	10%
Absent	457	27	95.2%	90%
Total	470	30	100	100

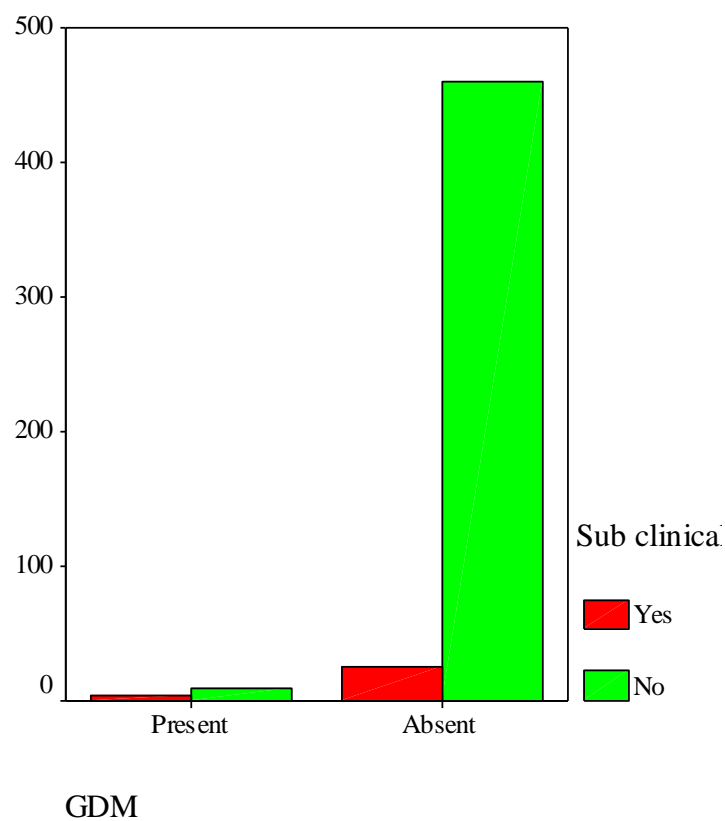
**The prevalence of GHTN in euthyroid women was
4.8% and 10% in SCH.**



PREVALENCE OF GDM IN Eu TH AND SCH

GDM	NUMBER		PERCENTAGE	
	EuTH	SCH	EuTH	SCH
Present	63	5	13.3%	16.6%
Absent	407	25	86.7%	84.4%
	470	30	100	100

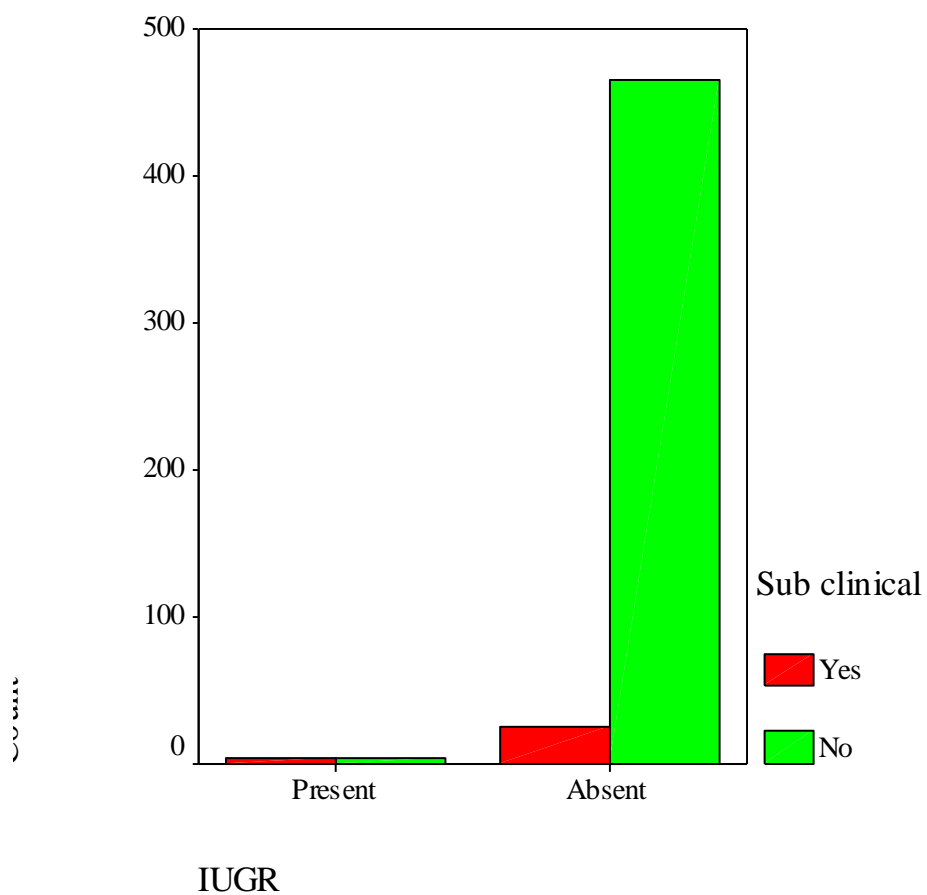
The prevalence of GDM in euthyroid and SCH women were 13.3%.vs16.6%



PREVALENCE OF IUGR IN EuTH AND SCH

IUGR	NUMBER		PERCENTAGE	
	EuTH	SCH	EuTH	SCH
Present	21	2	4.4%	6.6%
Absent	449	28	95.6%	93.4%
	470	30	100	100

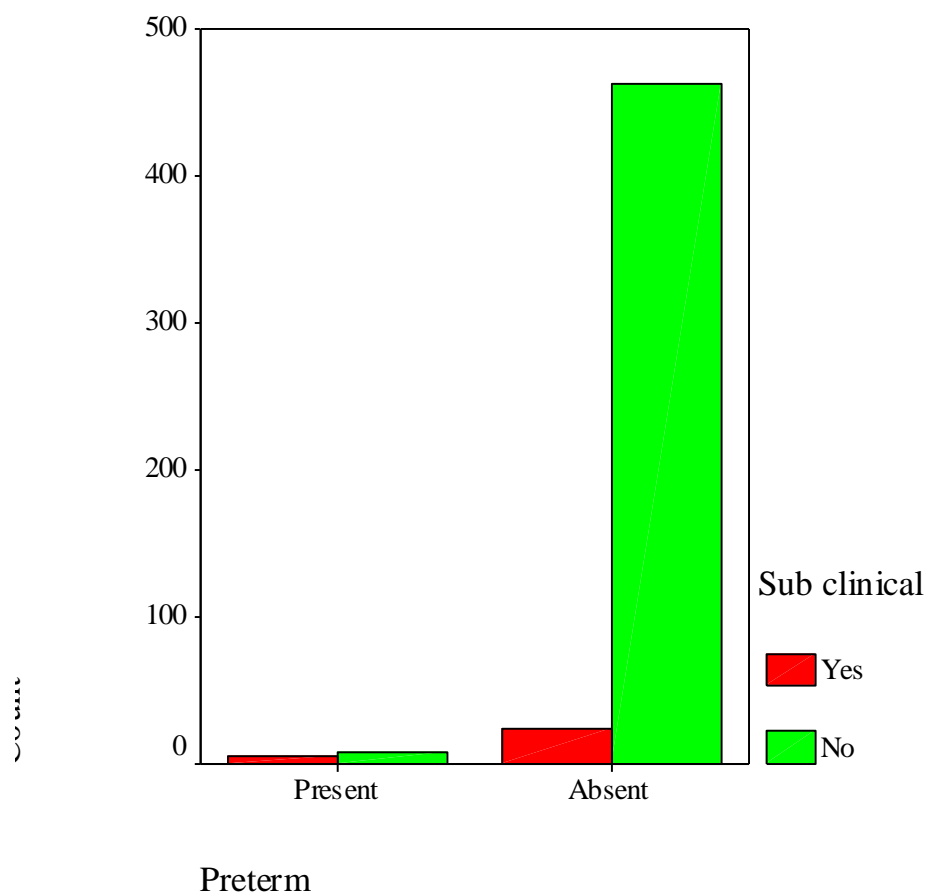
The prevalence of IUGR in euthyroid was 4.4% and 6.6% in SCH.



PREVALENCE OF PRETERM IN EuTH AND SCH

PRETERM	NUMBER		PERCENTAGE		P value
	EuTH	SCH	Eu TH	SCH	
Present	17	4	3.6%	13.3%	.031
Absent	453	26	96.4%	86.7%	
	470	30	100	100	

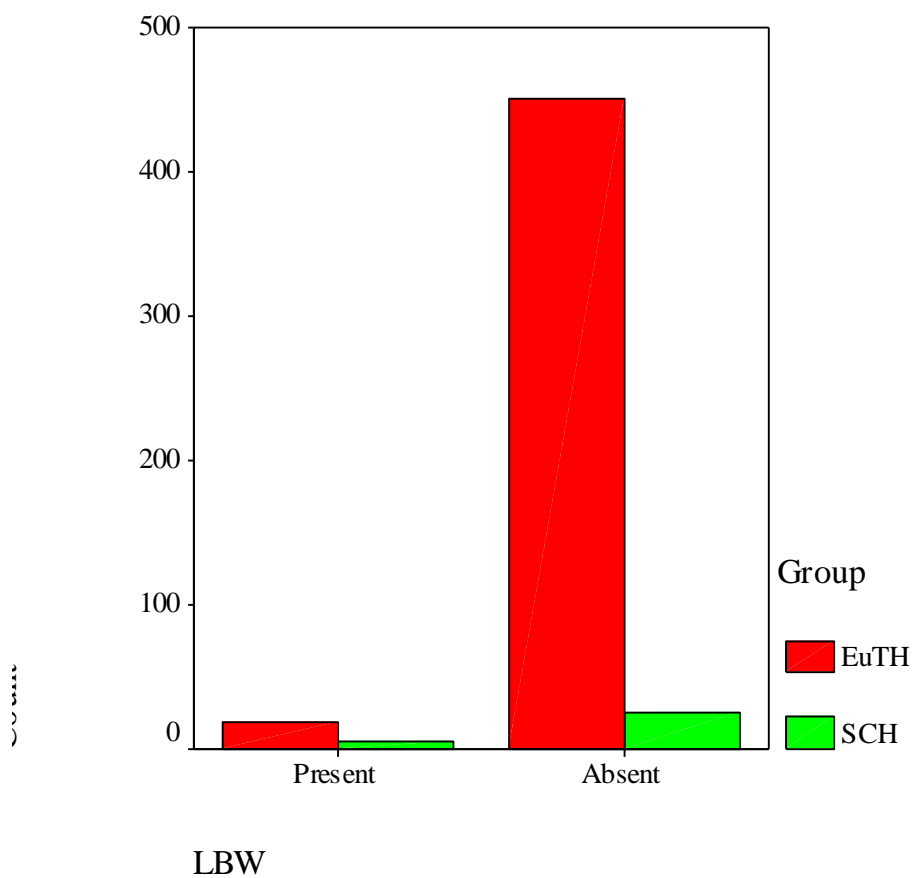
**The prevalence of preterm labour in euhyroid was
3.6% and 13.3% in SCH.**



PREVALENCE OF LOW BIRTH WEIGHT IN EuTH AND SCH

LOW BIRTH WT	NUMBER		PERCENTAGE		P value
	Eu TH	SCH	Eu TH	SCH	
Present	19	5	4.04%	23.5%	.010
Absent	464	25	95.96%	76.5%	
	470	30	100	100	

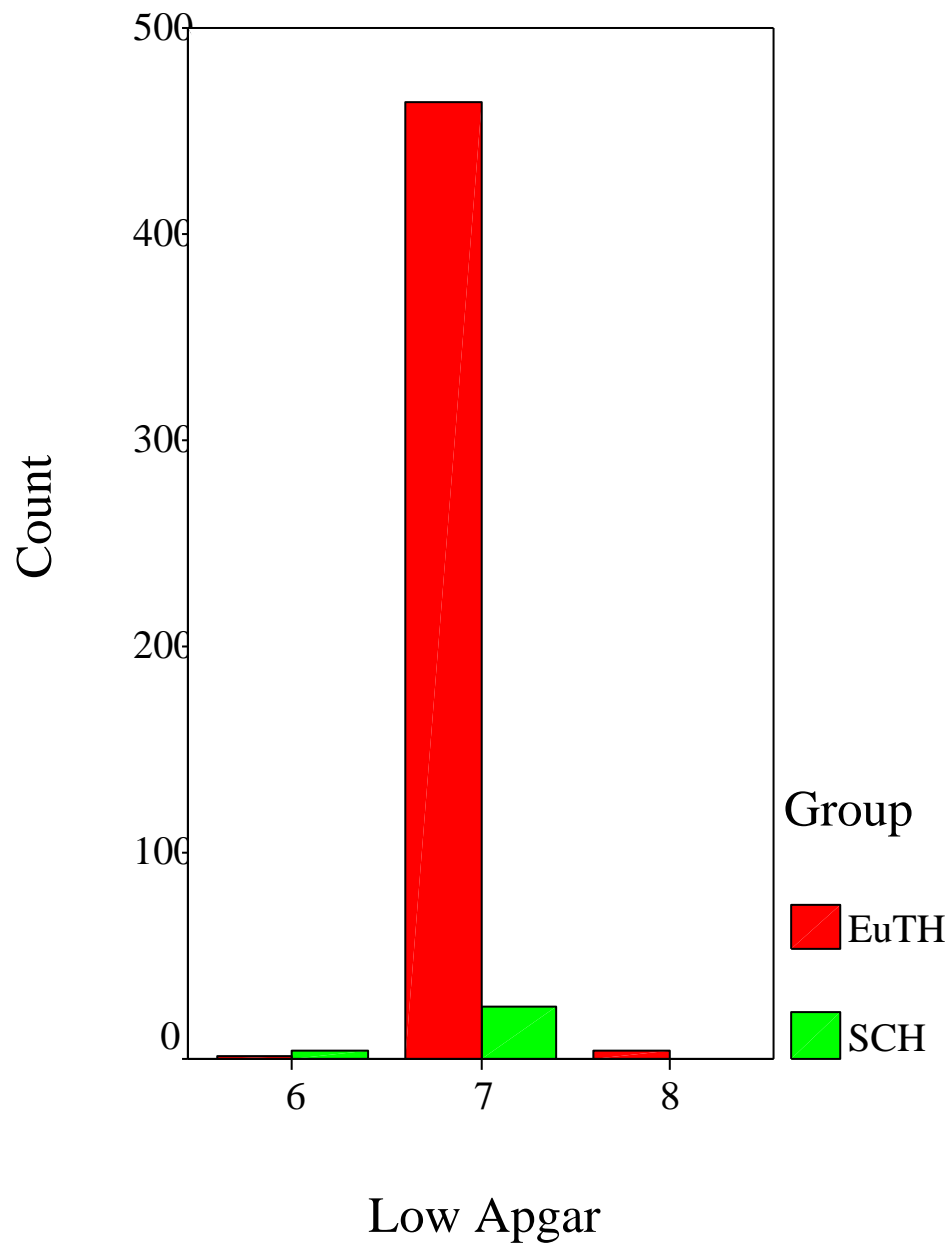
The prevalence of low birth weight in euthyroid women was 4.04% and 23.5% in SCH.



PREVALENCE OF LOW APGAR IN EuTH AND SCH

LOW APGAR	NUMBER		PERCENTAGE		P Value
	Eu TH	SCH	Eu TH	SCH	
6	2	4	0.4%	13.3%	
7	464	26	98.8%	86.7%	.000
8	4	0	0.8%	0	
Total	470	30	100	100	

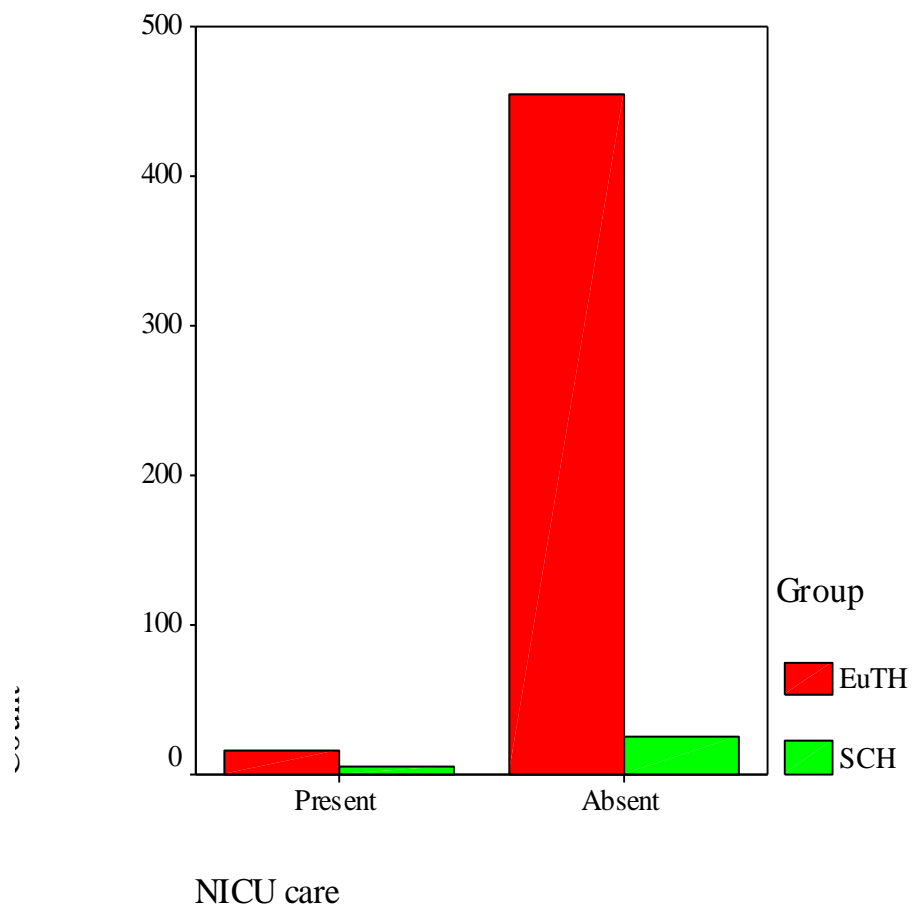
**The prevalence of low apgar in euthyroid women
was 0.4% and 13.3% in SCH**



PREVALENCE OF NICU CARE IN EuTH AND SCH

NICU CARE	NUMBER		PERCENTAGE		
	Eu TH	SCH	Eu TH	SCH	
Present	16	5	3.4%	16.6%	.006
Absent	454	25	96.6%	83.4%	
	470	30	100	100	

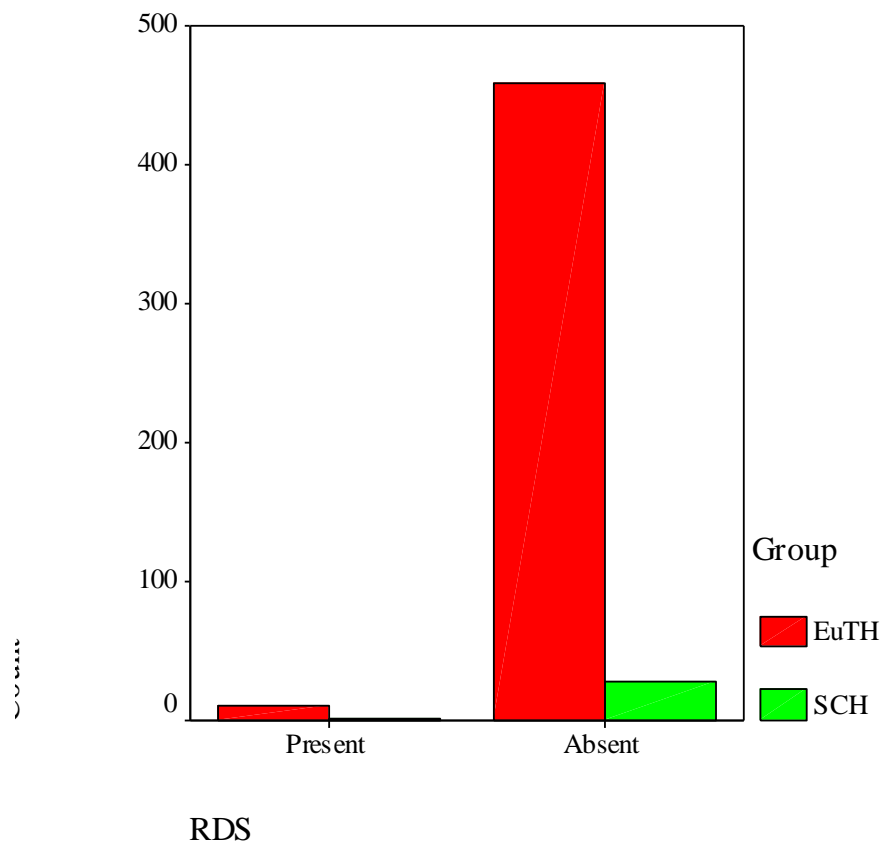
**The prevalence of NICU Care in euthyroid status
was 3.4% and 16.6%.**



PREVALENCE OF RDS IN Eu TH AND SCH

RDS	Number		Percentage		
	Eu TH	SCH	Eu TH	SCH	
Present	11	2	2.3%	6.6%	
Absent	459	28	97.7%	93.4%	.180
	470	30	100	100	

The prevalence of RDS in euthyroid was 2.3% and 6.6% in SCH.

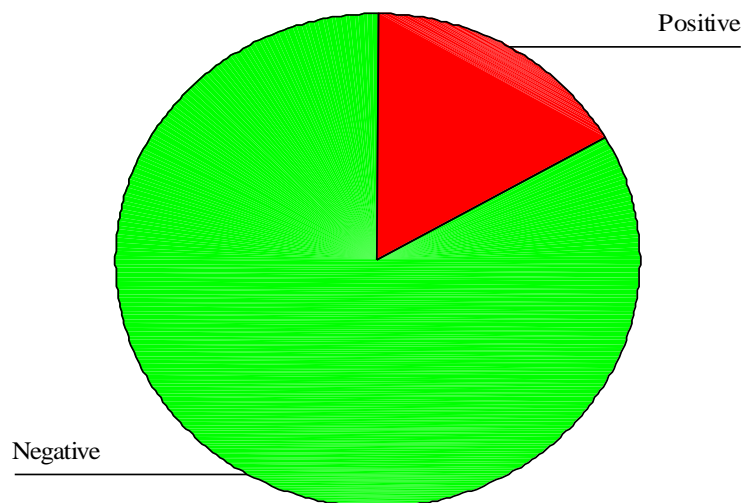


PREVALENCE OF TPO Ab POSITIVITY IN SCH

TPO Ab	Number	Percentage
Present	5	16.7%
Absent	25	83.3%
Total	30	100

Among the 30 SCH women, TPO Ab Positivity was 16.7%.

TPO Ab



DISCUSSION

Prevalence of subclinical hypothyroidism during pregnancy and its maternal and fetal outcomes study was conducted in Govt. R.S.R.M Lying in hospital from 2015 to 2016.

In this prospective study 500 women were enrolled .All subjects were screened observed and gave birth at the hospital .Thyroid function was tested at the first antenatal visit in Stanley hospital. Among 500 ,54.5 % were tested in first trimester, 29.6 % were tested in second trimester and 16 % were tested in third trimester. Information about the following demographic and clinical charecterstic were collected through proforma.

Fasting blood sample were collected in the morning from all subjects Serum TSH and ft4 concentration were measured by fully automated chemiluminescence immunoassay. The assessment of thyroid function based on the following trimester specific TSH and fT4according to the USPSTF Trimester specific values as per ATA2011, ES2012 and USPSTF GUIDELINES:

TRIMESTER	TSH(miu/dl)	fT4(ng/dl)
1 trimester	0.1-2.5	0.8-1.2
2 trimester	0.2-3	0.6-1.0
3 trimester	0.3-3	0.5-0.8

SCH defined as exceeding trimester specific TSH concentration and normal fT4.

Euthyroid defined as pregnant women with normal TSH and fT4 were considered euthyroid and served as control subjects.

All subject underwent regular antenatal checkup and delivered at govt. RSRM hospital.

The following maternal outcomes were diagnosed based on guidelines and documented .

GHTN was defined as SBP >140 mmhg and/ or DBP> 90mmhg after 20 weeks of pregnancy, with no previous history of hypertension, including preeclampsia.

Preeclampsia was defined as persistent elevated blood pressure with proteinuria.

GDM was defined as /or >140 mg/dl at 2 hr after 100mg OGCT. Preterm delivery defined as a delivery occurring between 28 and 37 weeks of gestation.

The following perinatal outcomes were assessed documented. IUGR was defined as an estimated fetal weight below the 10th percentile for gestation.

Fetal distress was defined as fetal heart rate 120bpm or 160bpm presence of meconium .LBW was defined as live birth weight 2500 g. RDS defined as ventilator support >24 hrs.

In this study , 500 pregnant women were screened out this 30 women was diagnosed as SCH. In our study prevalence of subclinical hypothyroidism is 6% Pregnant women with SCH referred to endocrinology OP at Stanley hospital to get adequate dose of L T4. They should be given treatment when TSH is above trimester specific values.

Patients with TSH values between 3-5mIU/ml were started on a dose of 25microgram and those with levels from 5-10mIU/ml were given 50microgram.

Based on whether they were started on treatment before 10 weeks and given prompt dosage titration, they were grouped as those receiving adequate treatment and inadequate treatment.

TSH levels were repeated for these patients 4-6 weeks after initiating the treatment or atleast in each trimester and thyroxine dosage titrated according to TSH.

A patient was considered to have received adequate treatment if the repeat TSH values were less than 3 mIU/ml Both the groups were followed till delivery and closely observed for the development of complications.

Among 30 SCH women, 13 were adequately treated and 17 in inadequately treated. Those who belong to inadequately treated group more prone for both maternal and perinatal adverse outcomes.

Maternal outcomes in the euthyroid and inadequately treated subclinical hypothyroidism group;

In our study more incidence of GHTN, pre eclampsia, preterm delivery in SCH compared to euthyroid women. (4.8 v 10 %, 3.4% v 9%, 3.6 v 13.3 %) which was similar to the outcomes of studies done by Wilson K L Casey B M et al 2007, Lieng mio chen et al 2014.

Studies of Manssota et al showed women with SCH had high incidence of pre eclampsia, and GHTN.

Similarly studies of Kharb et al.

Thyroid hormone have an effect on cardiovascular physiology and blood pressure regulation which are mediated by various molecular genomic mechanism that cause ventricular remodeling.

Studies have proven that thyroid hormone causes endothelial dysfunction, which characterised by decreased production of NO. It causes impaired vasorelaxation

Studies have also reported the altered function of liver and kidney during pre eclampsia causes decreased peripheral conversion of T4-T3. It causes T3 hypothyroxemia and increased TSH levels.

Loss of protein also causes hypothyroxemia.

Studies of wang et al and stagnaro green et al showed increased risk of preterm labour in pregnant women with SCH. Studies of cleary goldmann showed similar report.

IUGR

IUGR was not significantly high in SCH compared to euthyroid.

Studies of Idris et al, sahu et al and liang miao et al concluded high prevalence of iugr in women with SCH.

Studies of saki et al, pavarana et al showed high incidence of IUGR in women with SCH 13.7 %7.7% respectively.

Thyroid hormone is essential for growth and development of all vital organs. hypothyroid have negative effect on pituitary thyroid axis of newborn and interferes with normal vascular responsiveness and cardiovascular homeostasis of the fetus.

GDM

IN our study high prevalence of gdm 16.6% in women with SCH compared to 13.6% in euthyroid.

Studies of tudela et al showed 1,9-4.9% of women with SCH had GDM.

Studies of oliveria. agarwal et al, karkosta et al. Tizzo et al showed 16%,20.2%8.8% 10% of women with SCH developed GDM.

Studies of maratou et al has shown decreased rate of insulin stimulated glucose transport inside cells of hypothyroid women.

Increased insulin resistance was found in SCH women.

In our study, high incidence of Low birth weight(23.5% vs4.04%)p value of .001, low apgar at 5 min (13.3%vs1.4%) pvalue of .001, NICU admission (16.6% vs3.4%) p value of .001 in SCH group than euthyroid which was comparatively similar to the outcomes of studies done by Abalovich et al,Liang mio chen et al2014, zhongu fu et al2015 and J clinical endocrine metab 20 11 SCH has increase risk of fetal distress ,low birth weight, low apgar, nicu care and RDS.

In our study TPO Ab positivity about 16.7% in SCH group. which was similar to studies done by Bhattacharyya Rmukherjee et al 2012,Taka mastu et al.

SCH with TPO Ab positive will develop overt hypothyroidism within 2 yrs of delivery.

In our study SCH diagnosed about 56.7% in first trimester, 26.7% in second trimester and 16.6% in third trimester.

In our study 2 miscarriage in inadequately treated group.

In our study 47% of inadequately treated SCH group had high incidence of maternal complications than adequately treated group 7.6% which was similar to studies of Abolvich et al, negro et al .

In our study 32% of inadequately treated SCH group had high incidence of perinatal complications than adequately treated group 5%

SUMMARY

This includes 500 pregnant women belongs to 20-35yrs attending antenatal visit at R S R M Hospital.

We was screened at first antenatal visit with serum TSH and free T4 .Out of 500, 30 were diagnosed as subclinical hypothyroidism. In my study subclinical hypothyroidism is 6% prevalence, most of the SCH pregnant women were in the age group of 25-30yrs.

In this study, more than 56% women screened in first trimester.

Most of the SCH Women has TSH value in the range of 3.5-5miu/l.

There was 16% TPO Ab positive in women with SCH. There were 13 women belongs to adequately treated women with subclinical and has less incidence of complication than 17 inadequately treated SCH women

There were significant increased prevalence of about 9% Preeclampsia, 10% GHTN, 16.6% of GDM, 13.3% preterm birth compared to euthyroid pregnant women.

CONCLUSION

Thyroid hormone is essential for early placental development in pregnancy.

Especially during the first twelve weeks of pregnancy the fetus entirely depends upon the maternal thyroid hormone for the normal neural and skeletal development.

Hence early diagnosis and adequate treatment of maternal hypothyroidism in pregnancy is essential for decreasing the incidence of complications like miscarriage, preeclampsia, GHTN, preterm labour, low birth weight, low APGAR, NICU admission which are associated with subclinical hypothyroidism.

In our study 47% of inadequately treated group of subclinical hypothyroid women had high incidence of complication than adequately treated group 7.6%.

Inadequately treated group had a 3 fold increased risk of developing preeclampsia than treated group.

There was no significant increase in the incidence of abortion or fetal growth restriction in the inadequately treated group than euthyroid in our study.

The high incidence of GDM in our study is 16.6% than euthyroid

There was no case of placental abruption in my study group.

In our study inadequately treated group had high incidence of LBW, low APGAR, NICU admission, RDS than adequately treated group.

Adequately treatment group had significantly reduced incidence of perinatal complications 3.5% than 32% in inadequately treated group.

Hence, early screening, diagnosis and treatment will prevent maternal and fetal complications and reduces the complication of subclinical hypothyroidism in pregnancy.

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PROFORMA

DATE:

NAME:

AGE:

LMP:

IP NO:

EDD:

D.O.A:

D.O.D

OBSTETRIC COPE:

ADDRESS & CONTACT NO:

GESTATIONAL AGE:

PRESENTING COMPLAINTS:

ANY H/O SUGGESTIVE OF HYPO THYROIDISM

MENSTRUAL HISTORY:

MD SINCE:

OBSTETRIC HISTORY:

PAST HISTORY:

H/O HYPO THYROIDISM IN PREVIOUS PREGNANCIES

FAMILY H/O HYPO THYROIDISM

H/O HYPERTENSION/REAL DISORDERS / DIABETES / UTI

GENERAL :

EXAMINATION :

HEIGHT :

WEIGHT :

ANEMIA :

ICTERUS :

EDEMA :

PULSE :

BP :

CVS :

RS :

THYROID :

OBSTETRIC

EXAMINATION :

OGCT :

Hb

URINE MICROSCOPY:

THYROID FUNCTION TEST :

TPO Ab

CONSENT FORM

I agree to participate in the study entitled **‘PREVALENCE OF SUBCLINICAL HYPOTHYROIDISM IN PREGNANCY AND ITS MATERNAL AND FETAL OUTCOMES’**

I confirm that I have been told about this study in my mother tongue and have had the opportunity to clarify my doubts.

I understand that my participation is voluntary and I may refuse to participate at any time without giving any reasons and without affecting my benefits.

I agree not to restrict the use of any data or results that arise from this study.

Name of the participant

Sign /thumb print

Name of the investigator

Sign of the investigator

சுய ஒப்புதல் படிவம்

தைராய்டு சுரப்பியில் தைராய்டு ஹார்மோன் சுரப்பு தன்மை குறைந்துள்ளதை மகப்பேறு பெண்களுக்கு ஆய்வு மேற்கொள்ளுதல், மகப்பேறு மற்றும் பச்சிளங்குழந்தைகளுக்கு ஏற்படும் விளைவுகளை அறிதல் ஆய்வாளர் :

மரு. ஆர். மோனிகா

முதுநிலை பட்ட மேற்படிப்பு மாணவர்
மகப்பேறு மற்றும் பெண்கள் நலத்துறை
ஆர்.எஸ்.ஆர்.எம்.மருத்துவமனை
ஸ்டான்லி மருத்துவ கல்லூரி - சென்னை

பெயர் :

வயது :

உள்ளிருப்பு எண் :

இந்த மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது என்னுடைய சந்தேகங்களை தீர்க்கவும் அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டது

நான் இவ்வாய்வில் தன்னிச்சையாகத்தான் பங்கேற்கிறேன். எந்த காரணத்தினாலும் எந்த கட்டத்திலும் எந்த சட்டசிக்கலும் இன்றி இந்த ஆய்விலிருந்து விலகிக் கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.

நான் ஆய்விலிருந்து விலகிக்கொண்டாலும் ஆய்வாளர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கோ அல்லது உபயோகிக்கவோ என் அனுமதி தேவையில்லை எனவும் அறிந்து கொண்டேன் என்னை பற்றிய தகவல்கள் ரகசியமாக பாதுகாக்கப்படும் என்பதையும் அறிவேன்.

இந்த ஆய்வின் மூலம் கிடைக்கும் தகவல்களையும் பரிசோதனை முடிவுகளையும் ஆய்வாளர் அவர் விருப்பத்திற்கேற்ப பயன்படுத்திக் கொள்ளவும் அதனை பிரசுரிக்கவும் முழுமனதுடன் சம்மதிக்கிறேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன் எனக்கு கொடுக்கப்பட்டுள்ள அறிவுரைகளின்படி நடந்து கொள்வதுடன் ஆய்வாளருக்கு உண்மையுடன் இருப்பேன், என்றும் உறுதி அளிக்கிறேன்.

உடல்நலம் பாதிக்கப்பட்டாலோ வழக்கத்திற்கு மாறான ஏதேனும் நோய்குறி தென்பட்டாலோ அதனை தெரிவிப்பேன் என்றும் உறுதி கூறுகிறேன்.

இந்த ஆய்வில் எனக்கு எவ்விதமான பரிசோதனைகளையும் சிகிச்சைகளையும் மேற்கொள்ள நான் முழுமனதுடன் சம்மதிக்கிறேன்.

இப்படிக்கு,

ஆய்வாளரின் கையொப்பம்

நோயாளியின் கையொப்பம்

INSTITUTIONAL ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : Prevalence of subclinical Hypothodism in pregnancy
and its Masternal & Fetal outcome.

Principal Investigator : Dr. R Monica

Designation : PG, MS (O & G)

Department : Department of O & G
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 24.03.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.


MEMBER SECRETARY,
IEC, SMC, CHENNAI
MEMBER SECRETARY
ETHICAL COMMITTEE,
STANLEY MEDICAL COLLEGE
CHENNAI-600 001.

MASTER CHART

New.SNo	Sno	NAME	Sub_cli	Age	Trime	GA	TSH	FT4	TPOAb	OGCT	TREAT	mat_com	PRE ECLAMPSIA	GHTN	GDM	IUGR	OLIGO	PRETERM	FETAL DISTRESS	LABOUR COMPLICATION	mod_deli	APGAR	baby_wei	fet_com	LOW APGAR	LBW	PRETERM	NICU	RDS	TFT	DELAYED PASSAGE OF MECONIUM	PROLONGED NEONATAL JAUNDICE
31	1	*RENUKAPARMESWARI	1	30	2	37	3.86	1.00	2	170	1	GDM Anaemia	1	1	2	1	1	1	1	11	1	7	2.80	72 Hrs after birth TFT Normal	1	1	1	1	1	2	1	1
37	2	NANDHINI	1	28	1	10	3.10	1.07	2	94	2	Missed abortion	1								4				1	1	1	1	1	1	1	1
48	3	*KOKILA	1	29	1	38	5.30	1.04	2	77	1		1	1	1	1		1	1	1	1	7	2.60	72 Hrs after birth TFT Normal	1	1	1	1	2	1	2	1
74	5	*SHEELADEVI	1	30	1	36	4.25	1.08	2	91	2	GHTN,MSAF,Fetal distress	1	2	1	2		2	2	2	2	6	2.30	LBW NICU Admission,Low APGAR	2	2	1	2	1	2	1	1
80	6	*SIVAGAMI	1	26	3	38	3.55	1.00	2	87	2	GHTN,MSAF	1	2	1	1	2	1	1	2	3	6	2.80	2 Stage labour difficulty,low apgar	2	1	1	2	1	2	1	1
83	7	*CHITRA	1	29	1	37	3.69	0.90	2	119	1	oligo,PCOD, treated,induction completed	1	1	1	2	2	1	1	1	1	7	2.50	72 Hrs TFT WNL	1	1	1	1	1	2	1	1
93	8	*LIDYAJABASELVI	1	32	1	38	4.80	1.08	2	88	2	previous lscs GHTN	1	2	1	1		1	1	1	2	7	2.95	72 Hrs TFT WNL	1	1	1	1	1	2	1	1
100	9	*TEJASRI	1	28	1	37	5.10	1.01	2	158	2	Oligo,CPD,infertility Rxed,GDM	1	1	2	1	2	1	1	2	2	6	2.60	Low APGAR,NICU Admission	1	1	1	1	1	2	1	1
108	10	*YASMINFATHIMA	1	22	2	38	3.50	1.20	2	90	1		1	1	1	1		1	1	1	1	7	2.90	72 hrs TFT WNL	1	1	1	1	1	2	1	1
116	11	*REBAKA	1	30	1	38	4.26	1.07	2	110	1	previous lscs	1	1	1	1	1	1	1	1	2	7	2.70	72 Hrs TFT WNL	1	1	1	1	1	2	1	1
130	12	*GOWSAR	1	28	1	37	4.80	1.20	2	116	2	Anaemia,Fetal distress	1	1	1	1	1	1	2	1	2	7	2.80	72 Hrs TFT WNL	1	1	1	1	1	2	1	1
171	13	*MUNNI	1	30	3	37	3.70	1.03	2	109	2	GHTN	1	2	1	1	1	1	1	1	2	7	2.80	72Hrs TFT WNL	1	1	1	1	1	2	1	1
201	14	*ESTHERSHEEBA	1	31	2	38	4.04	1.09	2	112	1		1	1	1	1	1	1	1	1	1	7	2.80	72 hrs TFT WNL	1	1	1	1	1	2	1	1
262	15	*GOMATHY/nagaraj	1	24	1	37	4.51	1.10	2	92	1		1	1	1	2	1	1	1	1	1	7	2.80	72 Hrs TFT WNL	1	1	1	1	1	2	1	1
280	16	*SANGEETHA/Vetrivel	1	32	3	36	4.70	0.90	1	89	1		1	1	1	1	1	2	2	2	3	6	2.25	Low APGAR,Late preterm,NICU admission	2	2	2	2	2	2	1	1
290	17	*SHAIDHABANU	1	32	2	37	4.80	1.10	2	82	1		1	1	1	1	1	1	1	1	2	7	2.50	TOF Anomaly baby,NICU admission	1	1	2	2	2	2	1	1
303	18	*AMSAVENI	1	29	1	39	3.98	1.04	2	88	1		1	1	1	1	1	1	1	1	1	7	3.20	72 Hrs TFT WNL	1	1	1	1	1	2	1	1
312	19	*DIVYA/Jeyaseelan	1	29	2	37	4.90	1.10	1	116	2	previous lscs	2	1	1	1	2	2	1	1	2	7	2.40	NICU Admission,LBW	1	2	1	2	1	2	1	1
334	20	*SHANTHI/Sivanatham	1	28	1	39	3.65	1.05	2	106	1		1	1	1	1	1	1	1	1	1	7	2.90	72Hrs TFT WNL	1	1	1	1	1	2	1	1
357	21	*KALAISELVI	1	28	2	37	3.80	1.00	2	170	1	GDM	2	1	2	1	1	1	1	1	1	7	2.90	NICU Admission	1	1	1		1		1	1
394	22	SANGEETHA/Parthiban	1	22	3	37	3.65	0.8	2	85	2	fetal distress	1	1	1	1	1	1	2	1	2	6	2.43	LBW/NICU Admission,low APGAR	2	2	2	2	2	2	1	1
420	23	ELAKIYIA/Yuraj	1	24	1	37	4.12	1.30	2	93	2	2nd stage delay	1	1	1	1	1	1	1	2	3	7	2.80	NICU Admission,72 hrs TFT WNL	1	1	1	1	1	2	1	1
432	24	ANITHA	1	32	2	37	5.30	1.07	1	164	2	Overt GDM,Previous lscs	1	1	2	1	1	1	1	1	2	7	3.50	NICU observation,72hrs TFT WNL	1	1	1	1	1	2	1	1
446	25	KASTURI	1	30	1	37	3.75	1.00	2	106	2	GHTN	1	2	1	1	1	1	1	1	2	6	2.75	NICU Admission,Low APGAR	2	1	1	2	1	2	1	1
455	26	POOVIZHI	1	29	1	38	4.03	1.1	2	87	1		1	1	1	1	1	1	1	1	1	7	3.00	72 Hrs TFT WNL	1	1	1	1	1	2	1	1
477	477	KAVITHA/Pannerselvam	1	30	3	37	3.70	0.90	2	110	2	Failed induction,preeclampsia	1	1	1	1	1	1	1	1	2	7	2.80	72Hrs TFT WNL	1	1	1	1	1	2	1	1
485	485	REVATHY	1	30	3	35	3.80	1.02	2	98	2	IUGR,Oligo,Preterm	2	1	1	1		2	1	2	1	7	2.40	LBW,Late Preterm	1	2	2	1	1	2	1	1
489	29	SONIYA	1	25	1	33	3.90	1.14	1	93	2	preterm	1	2	1	2	2	2	1	1	1	7	2.10	NICU Admission,LBW,Preterm	1	2	2	2	1	2	1	1
499	499	VAIDHIGEI	1	24	1	10	3.70	1.03	1	102	2		1	2							4				1	1	1	1	1	1	1	1
500	500	BHAVANI	1	21	3	33	3.80	1.05	2	94	2	GHTN,ABRUPTIO PLACENTA	1	2	1			2	1	1	2	6	1.90	NICU,PRETERM,LBW,Low APGAR	2	2	2	2	1	2	1	1
1	1	Meena	2	30	1	39	0.90	1.09		110											1	7	3.00								1	
2	2	Elakiya	2	26	1	37	2.90	1.01		78		CPD									2	7	2.90									
3	3	Devi	2	28	1	37	2.10	1.00		92											1	8	2.72									
4	4	komala	2	30	1	37	1.20	0.90		108		Rh-ve									1	7	3.00									

[illegible]

[illegible]

New.SNo	Sno	NAME	Sub_cli	Age	Trime	GA	TSH	FT4	TPOAb	OGCT	TREAT	mat_com	PRE ECLAMPSIA	GHTN	GDM	IUGR	OLIGO	PRETERM	FETAL DISTRESS	LABOUR COMPLICATION	mod_deli	APGAR	baby_wei	fet_com	LOW APGAR	LBW	PRETERM	NICU	RDS	TFT	DELAYED PASSAGE OF MECONIUM	PROLONGED NEONATAL JAUNDICE
79		ponni	2	21	1	38	1.10	1.01		125											1	7	2.90									
81		Mariyam fathima	2	24	2	38	1.72	1.01		98		MSAF									2	7	3.10									
82		Gomathi	2	24	1	39	1.60	1.18		90		Fetal distress							2		2	7	3.50									
84		Desarani	2	25	1	38	0.60	1.09		89		previous lscs									2	7	2.80									
85		kalpana	2	25	1	37	0.80	1.50		145		GDM									1	7	3.00									
86		Shalini	2	23	1	39	2.90	1.10		78					2						1	7	2.90									
87		Priya	2	22	2	38	2.70	1.20		90											1	7	2.90									
88		Revathi	2	24	2	39	2.26	1.06		94		Anaemia									1	7	2.70									
89		Nandhini	2	29	1	37	2.10	1.40		106											1	7	2.50									
90		Gandhimathi	2	27	1	38	2.33	1.05		82											1	7	2.80									
91		Deepa	2	29	2	38	1.30	1.03		90		previous lscs									2	7	3.00									
92		Shameema	2	21	3	39	2.21	1.05		76		Non reactive CTG									2	7	3.20									
94		Valliammal	2	28	1	39	2.01	1.07		95											1	7	2.80									
95		Sathya	2	24	1	37	2.00	1.20		91											1	7	2.75									
96		Geethalakshmi	2	27	2	38	2.20	1.09		99											1	7	2.90									
97		Rihanna	2	21	2	38	2.10	1.01		80		previous lscs									2	7	3.00									
98		Divyabharathi	2	25	2	39	1.30	1.01		79		Failure to progress									2	7	3.10									
99		Shanthi	2	25	1	38	1.50	1.07		91		previous lscs									2	7	3.50									
101		Archana	2	23	1	39	2.00	1.05		89											1	7	3.00									
102		Vasanthi	2	25	2	39	0.80	1.30		103											1	7	2.90									
103		Rashikamehar	2	25	1	38	2.30	1.10		120											1	7	2.75									
104		Kalarani	2	26	3	37	0.90	1.20		78											1	7	2.60									
105		Vidhyarani	2	24	3	37	1.20	1.03		166		previous lscs			2						2	7	2.55									
106		Rangeela	2	23	1	38	1.90	1.50		112		CPD									2	7	3.10									
107		Suryia	2	23	1	38	2.10	1.06		97		previous lscs									2	7	3.00									
109		Kanmani	2	21	1	37	0.70	1.00		94											1	7	2.75									
110		Dolphinros	2	27	1	38	1.73	1.30		118		previous lscs									2	7	3.00									
111		Annapoorani	2	25	2	38	2.80	1.08		121		previous lscs									2	7	3.20									
112		Rajalakshmi	2	22	2	38	1.50	1.00		131		previous lscs									2	7	3.60									
113		Dhanalakshmi	2	29	2	37	2.60	1.50		90		Fetaldistress							2		2	7	3.00									
114		Jeyasiili	2	28	2	37	2.10	1.20		86											1	7	2.60									
115		Gowsiya	2	29	1	38	0.90	1.40		74		previous lscs									2	7	3.00									
117		Radha	2	29	1	37	1.30	1.01		105		previous lscs									2	7	3.90									
118		Gomathy	2	24	3	39	2.00	1.05		91		Fetal distress							2		2	7	3.40									
119		Muthulakshmi	2	26	1	39	1.10	1.01		89		Failed induction									2	7	2.95									

New.SNo	Sno	NAME	Sub_cli	Age	Trime	GA	TSH	FT4	TPOAb	OGCT	TREAT	mat_com	PRE ECLAMPSIA	GHTN	GDM	IUGR	OLIGO	PRETERM	FETAL DISTRESS	LABOUR COMPLICATION	mod_deli	APGAR	baby_wei	fet_com	LOW APGAR	LBW	PRETERM	NICU	RDS	TFT	DELAYED PASSAGE OF MECONIUM	PROLONGED NEONATAL JAUNDICE
120		Punitha	2	30	2	37	2.30	1.07		85		GHTN,Failed induction		2							2	7	3.00									
121		Bhavani	2	28	3	39	2.10	1.02		79		Fetaldistress							2		2	7	3.20									
122		Rathi	2	28	1	38	2.00	1.10		107											1	7	2.98									
123		Sona	2	27	2	39	0.50	1.00		102		Rh-ve previous LSCS									2	7	3.00									
124		Rohnni	2	23	2	39	1.70	1.09		96		CPD									2	7	3.40									
125		Shruthi	2	21	3	38	0.80	1.20		92		previous lscs									2	7	3.00									
126		Fathima	2	28	1	37	1.20	1.01		88											1	7	3.00									
127		Suganya	2	25	1	38	1.70	1.08		82											1	7	2.70									
128		Indumathi	2	25	2	37	1.27	1.50		94											1	7	2.70									
129		Anusya	2	22	3	37	2.70	1.40		158					2						1	7	2.50									
131		Devipriya	2	21	1	37	1.90	1.20		123											1	7	2.90									
132		kavitha	2	21	2	37	2.20	1.09		100		Oligo					2				2	7	3.00									
133		Sowjana	2	24	3	39	2.40	1.40		90		IUGR				2					2	7	2.50									
134		Sabeera	2	25	1	40	2.10	1.10		119											1	7	3.00									
135		Shanthi	2	21	2	38	1.80	1.10		99											1	7	2.80									
136		Nagalakshmi	2	23	2	38	2.60	1.30		86		previous lscs									2	7	3.70									
137		Sudha	2	23	1	39	2.30	1.20		92											1	7	3.00									
138		Kowslya	2	24	1	37	2.70	1.10		79		previous lscs									2	7	2.95									
139		Reshma	2	25	1	38	0.70	1.00		101											1	7	2.75									
140		Bhuvana	2	28	2	37	0.40	1.00		109		Oligo					2				2	7	2.78									
141		Sampooranam	2	29	1	37	0.40	1.20		102											1	7	2.67									
142		Keerthipriya	2	23	3	39	1.00	1.20		96											1	7	2.45									
143		Nishanthini	2	28	2	37	1.84	1.30		83											1	7	2.50									
144		Indumathi	2	27	1	37	2.40	1.15		77											1	7	2.80									
145		kalpana	2	27	1	38	1.20	1.00		89		previous lscs									2	7	2.80									
146		Deepa	2	27	1	37	1.30	1.40		91		previous lscs									2	7	3.00									
147		Kalaiselvi	2	32	1	39	2.60	1.20		97		Failed induction									2	7	3.20									
148		Nandhini	2	27	2	38	2.02	1.05		104		Failure to progress									2	7	3.50									
149		Divya	2	28	1	38	2.30	1.04		113		previous lscs									2	7	3.60									
150		Hemalatha	2	26	1	38	1.90	1.04		124		previous lscs									2	7	3.00									
151		Soniya	2	24	2	37	2.40	1.10		105		MS AF,Fetal distress							2		2	7	2.80									
152		Vadiheyi	2	24	2	37	2.10	1.06		92		Rh-ve									1	7	2.60									
153		Munniyammal	2	28	1	37	2.90	1.01		96											1	7	2.90									
154		Lavanya	2	25	2	37	2.28	1.01		89											1	7	2.70									
155		Srirathi	2	27	1	38	0.60	1.01		86											1	7	2.70									

New.SNo	Sno	NAME	Sub_cli	Age	Trime	GA	TSH	FT4	TPOAb	OGCT	TREAT	mat_com	PRE ECLAMPSIA	GHTN	GDM	IUGR	OLIGO	PRETERM	FETAL DISTRESS	LABOUR COMPLICATION	mod_deli	APGAR	baby_wei	fet_com	LOW APGAR	LBW	PRETERM	NICU	RDS	TFT	DELAYED PASSAGE OF MECONIUM	PROLONGED NEONATAL JAUNDICE
192		Vaishali	2	23	1	37	1.40	1.03		84		short primi									2	7	2.50									
193		Deepika	2	26	1	38	1.20	1.08		109		previous lscs									2	7	2.80									
194		Aruna	2	21	1	39	2.50	1.07		111											1	7	3.00									
195		Stella	2	25	2	37	1.30	1.09		123											1	7	2.80									
196		Geetha	2	25	1	38	2.50	1.30		89											1	7	2.70									
197		Uma	2	27	3	37	0.40	1.10		78		Anaemia									1	7	2.30									
198		Pradeepa	2	21	1	39	2.00	1.02		94		Primi breech									2	7	2.80									
199		Meharsulthana	2	23	1	40	2.20	1.10		90		previous lscs									2	7	3.10									
200		Saritha	2	21	1	39	1.60	1.20		107											1	7	3.00									
202		Arummaikannu	2	27	1	37	1.40	1.10		105		previous lscs									2	7	3.00									
203		Muneeswari	2	25	3	37	2.13	1.09		86		Severe oligo					2				2	7	2.90									
204		Padma	2	24	1	38	1.54	1.07		165		CPD			2						2	7	2.89									
205		Chinnaponnu	2	25	1	37	1.65	1.01		88											1	7	2.60									
206		Pongathai	2	25	1	37	0.60	1.02		74		GHTN		2							1	7	2.70									
207		Ambiga	2	23	2	37	2.10	1.40		94		Anaemia									1	7	2.50									
208		Baseera	2	23	1	37	2.80	1.06		98											1	7	2.70									
209		Mubeena	2	23	2	37	2.30	1.10		106		preclampsia abruption	2								2	7	2.70									
210		Hema	2	21	2	37	1.20	1.08		115		previous lscs									2	7	2.70									
211		Nithya	2	22	1	38	0.30	1.80		93											1	7	2.90									
212		Gowthami	2	24	1	39	2.70	1.07		117											1	7	3.00									
213		sulochanna	2	24	1	40	1.49	1.02		112											1	7	3.10									
214		Vanja	2	25	3	37	1.35	1.02		132		MSAF,Fetal distress									2	7	3.00									
215		Hidha	2	23	2	38	2.31	1.07		85											1	7	2.75									
216		Malliga	2	29	1	39	1.63	1.01		90		previous lscs									2	7	2.90									
217		Vijalakshmi	2	27	2	37	1.14	1.10		92											1	7	2.80									
218		Divya	2	25	1	38	2.13	1.04		79		previous lscs									2	7	3.00									
219		Rajalakshmi	2	30	2	37	2.02	1.01		103											1	7	2.50									
220		Brindha	2	32	1	38	2.20	1.02		116		previous lscs									2	7	2.80									
221		Akila	2	31	1	37	1.32	1.06		121											1	7	2.50									
222		Ezhil	2	29	1	37	1.74	1.20		126		short primi									2	7	2.90									
223		Pankajam	2	28	1	40	1.63	1.05		102		Primi breech									2	7	3.00									
224		Alamelu	2	28	1	39	2.10	1.10		89											1	7	3.10									
225		Sinduja	2	27	2	37	1.01	1.50		96											1	7	3.00									
226		Poornima	2	25	3	38	0.80	1.25		92		previous lscs									2	7	2.75									
227		Valli	2	26	3	38	1.20	1.40		78		previous lscs									2	7	2.80									

New.SNo	Sno	NAME	Sub_cli	Age	Trime	GA	TSH	FT4	TPOAb	OGCT	TREAT	mat_com	PRE ECLAMPSIA	GHTN	GDM	IUGR	OLIGO	PRETERM	FETAL DISTRESS	LABOUR COMPLICATION	mod_deli	APGAR	baby_wei	fet_com	LOW APGAR	LBW	PRETERM	NICU	RDS	TFT	DELAYED PASSAGE OF MECONIUM	PROLONGED NEONATAL JAUNDICE
228		Sumathi	2	25	1	37	1.88	1.03		90											1	7	2.60									
229		Preethi	2	23	1	37	1.56	1.09		104											1	7	2.50									
230		Jaya	2	25	2	40	0.70	1.10		87		Failed induction									2	7	2.80									
231		Sathya	2	24	2	38	2.20	1.06		120		previous lscs									2	7	3.00									
232		Sivaranjani	2	25	1	38	1.40	1.05		110		previous lscs									2	7	3.00									
233		Vedhanayagi	2	21	1	37	1.60	1.01		108		previous lscs									2	7	2.90									
234		Kalliammal	2	26	1	37	2.00	1.02		98											1	7	2.80									
235		Lekha	2	21	1	34	1.50	1.02		93								2			1	7	2.00	NICU								
236		Vannathy	2	22	2	38	0.90	1.30		91											1	7	2.70									
237		Kalaivani	2	23	2	37	1.10	1.08		89											1	7	2.70									
238		Radhika	2	27	3	38	2.10	1.05		160		CPD			2						2	7	3.00									
239		Rani	2	29	3	39	1.40	1.07		105		Fetal distress									2	7	3.30									
240		Shakila	2	22	2	37	1.92	1.20		102											1	7	3.00									
241		venkatalakshmi	2	25	1	38	2.00	1.01		112		previous lscs									2	7	3.25									
242		Ponny	2	27	2	37	2.80	1.09		88											1	7	3.10									
243		Yavaneswari	2	28	2	38	1.20	1.07		90		Rh-ve previous LSCS									2	7	3.45									
244		Sarojini	2	23	1	37	1.50	1.03		95											1	7	2.76									
245		Roja	2	23	1	38	2.30	0.90		99											1	7	2.60									
246		Chandra	2	22	2	38	0.60	1.20		114											1	7	2.50									
247		Bhavani	2	26	1	39	1.39	1.07		100											1	7	2.90									
248		Shanu	2	30	2	38	1.10	1.03		94		Failure to progress									2	7	3.00									
249		Jaishini	2	29	1	37	1.26	1.01		90		previous lscs									2	7	3.00									
250		Elakiyaselvi	2	21	1	39	1.40	1.01		89											1	7	2.90									
251		Tamilarasi	2	19	1	40	2.10	1.09		78											1	7	2.80									
252		Kamala	2	21	2	37	1.45	1.02		86		Anaemia									1	7	2.50									
253		vinodhini	2	27	1	37	1.20	1.01		110											1	7	2.80									
254		Punitha	2	27	1	38	1.00	1.05		93		previous lscs									2	7	3.00									
255		Ramanidevi	2	24	2	37	2.50	1.07		86											1	7	2.70									
256		Ayeshabanu	2	27	1	35	1.80	1.07		82								2			1	7	2.10	NICU								
257		Subbulakshmi	2	31	2	38	1.69	1.01		90		previous lscs									2	7	3.00									
258		Leelapriya	2	25	1	38	1.23	1.10		102		previous lscs									2	7	3.50									
259		Joysmary	2	25	2	39	0.80	1.01		95		Failed induction									2	7	3.20									
260		Maniarasi	2	22	1	40	1.75	1.05		107											1	7	3.00									
261		Rathinakumari	2	21	3	38	0.50	0.90		96		previous lscs									2	7	2.90									
263		vishnupriya	2	25	2	38	1.09	1.01		90											1	7	2.75									

New.SNo	Sno	NAME	Sub_cli	Age	Trime	GA	TSH	FT4	TPOAb	OGCT	TREAT	mat_com	PRE ECLAMPSIA	GHTN	GDM	IUGR	OLIGO	PRETERM	FETAL DISTRESS	LABOUR COMPLICATION	mod_deli	APGAR	baby_wei	fet_com	LOW APGAR	LBW	PRETERM	NICU	RDS	TFT	DELAYED PASSAGE OF MECONIUM	PROLONGED NEONATAL JAUNDICE
264		Ellamparuthi	2	31	3	37	1.20	1.16		89		preclampsia,failed induction	2								2	7	3.00									
265		kalyani	2	23	3	37	0.40	1.63		78		previous lscs									2	7	3.10									
266		Srimeenatchi	2	30	2	37	1.34	1.20		83		Anaemia									1	7	2.50									
267		Vidhya	2	31	1	39	2.23	1.19		90											1	7	2.80									
268		Sasipriya	2	29	1	40	1.27	1.03		94											1	7	2.75									
269		Sowmiya	2	25	1	38	1.36	1.27		88		previous lscs									2	7	3.30									
270		Santhalakshmi	2	28	2	39	1.72	0.90		96											1	7	3.00									
271		Christy	2	27	1	39	1.60	1.08		105		previous lscs									2	7	3.20									
272		Vanitha	2	25	1	38	2.40	1.12		86		previous lscs									2	7	3.50									
273		Banumathi	2	26	1	37	1.25	1.53		89											1	7	2.90									
274		Priyadharshini	2	21	2	39	2.30	1.06		110											1	7	2.75									
275		Divya	2	23	3	40	1.84	1.00		104		Fetal distress									2	7	3.00									
276		Jammuna	2	27	2	39	2.02	1.17		101		Non reactive CTG									2	7	2.75									
277		srikala	2	22	2	39	1.44	1.02		86											1	7	2.65									
278		Jayarani	2	21	1	37	2.80	1.11		92											1	7	2.55									
279		Astalakshmi	2	24	1	37	1.39	1.09		95											1	7	2.82									
281		Ganeswari	2	26	2	34	1.99	1.01		76								2			2	7	1.90	NICU								
282		Madhavi	2	28	2	38	1.30	1.26		90		previous lscs									2	7	3.05									
283		Shenbagavalli	2	27	1	39	1.34	1.17		94		previous lscs									2	7	3.20									
284		Vimala	2	30	1	38	1.00	1.45		92		previous lscs									2	7	3.10									
285		Kalpana	2	29	1	40	0.70	1.81		104											1	7	2.90									
286		Meena	2	31	1	39	1.47	1.05		101											1	7	2.75									
287		Sasikala	2	24	3	37	1.85	1.13		96		Anaemia									1	7	2.60									
288		Gayathri	2	29	1	38	1.56	1.68		92											1	7	2.75									
289		Rajeswari	2	26	3	37	1.10	1.04		89		GHTN,Failed induction		2							2	7	2.80									
291		kavitha	2	24	2	37	1.21	0.80		78		previous lscs									2	7	2.75									
292		Muthuselvi	2	26	1	39	1.35	1.02		87		previous lscs									2	7	3.00									
293		Sujanthini	2	28	1	40	1.61	1.09		91		CPD									2	7	3.20									
294		Padmapriya	2	25	2	40	1.71	1.15		98		Fetal distress									2	7	3.50									
295		Amsaveni	2	27	1	39	1.63	1.23		95											1	7	2.90									
296		Raseetha	2	30	2	39	1.02	1.50		103											1	7	2.75									
297		Vennila	2	27	1	38	2.34	1.03		107											1	7	2.65									
298		Nirajannadevi	2	26	2	37	1.41	1.01		94											1	7	2.80									
299		Thirumangai	2	29	1	39	2.01	1.07		110		previous lscs									2	7	3.00									
300		Kaviya	2	21	3	35	1.89	1.19		89		severe oligo									2	7	2.20									

[illegible]

New.SNo	Sno	NAME	Sub_cli	Age	Trime	GA	TSH	FT4	TPOAb	OGCT	TREAT	mat_com	PRE ECLAMPSIA	GHTN	GDM	IUGR	OLIGO	PRETERM	FETAL DISTRESS	LABOUR COMPLICATION	mod_deli	APGAR	baby_wei	fet_com	LOW APGAR	LBW	PRETERM	NICU	RDS	TFT	DELAYED PASSAGE OF MECONIUM	PROLONGED NEONATAL JAUNDICE
339		Ambiga	2	22	3	39	1.09	1.06		92		Fetal distress							2		2	7	2.90									
340		priyadharshini	2	23	2	40	1.40	1.21		120		CPD									2	7	3.00									
341		Akila	2	24	3	37	2.50	1.03		108		previous lscs									2	7	3.00									
342		Reshmi	2	27	1	38	1.70	1.09		112											1	7	2.70									
343		Anbumalar	2	29	1	39	1.40	1.18		94		short primi									2	7	3.00									
344		kalaimathi	2	28	1	38	1.20	1.45		92		previous lscs									2	7	3.00									
345		Deepthi	2	27	3	37	1.39	1.01		89		Transverse lie									2	7	2.75									
346		Nisha	2	20	1	38	2.00	1.07		76		previous lscs									2	7	3.00									
347		Pappathy	2	25	2	40	1.11	1.05		119											1	7	3.10									
348		Salomi	2	28	3	37	1.94	1.12		95											1	7	2.60									
349		Umadevi	2	23	3	37	1.54	1.00		103								2			1	7	1.80									
350		Swathi	2	21	3	37	1.31	1.20		88											1	7	2.50									
351		Durga	2	23	1	39	1.68	1.40		90											1	7	2.80									
352		Bharathi	2	24	2	37	2.71	1.03		102		preclampsia,failed induction	2								2	7	2.75									
353		Hemaakilandeswari	2	28	2	38	1.20	1.07		94		GDM CPD			2						2	7	3.60									
354		Radika	2	21	1	38	1.17	1.09		85		previous lscs									2	7	3.50									
355		Manimegalai	2	23	1	38	1.28	1.29		88		previous lscs									2	7	3.00									
356		Rangeela	2	27	3	37	1.35	1.17		92		severe oligo									2	7	2.20									
358		karpagam	2	21	3	37	2.30	0.90		101											1	7	2.70									
359		Deni	2	30	2	37	1.19	1.31		117											1	7	2.70									
360		shanthi	2	27	1	37	1.22	1.01		120											1	7	2.50									
361		Suganya	2	25	3	37	1.90	1.26		86											1	7	2.50									
362		Shymala	2	23	2	37	2.04	1.07		90											1	7	2.60									
363		Divyabharathi	2	23	3	38	1.71	1.15		94		MSAF,Fetal distress							2		2	7	3.00									
364		Ammulu	2	36	2	37	1.08	1.21		105											1	7	2.70									
365		Sheeba	2	24	3	37	1.79	1.20		90											1	7	2.75									
366		Hafia	2	22	3	37	1.13	1.10		96											1	7	2.50									
367		Cyindhiya	2	21	3	37	1.05	1.05		92											1	7	2.50									
368		Rahini	2	25	1	39	1.82	1.12		100											1	7	3.00									
369		Kalliammal	2	25	2	37	0.50	1.50		86		GHTN		2							1	7	2.80									
370		Neela	2	32	1	38	2.27	1.41		79											1	7	2.30									
371		Ganga	2	29	1	39	1.25	1.02		82											1	7	2.50									
372		Ellammal	2	26	1	37	1.47	1.18		94											1	7	3.00									
373		Suba	2	28	1	37	2.39	1.25		107											1	7	2.70									
374		Lakshmipriy	2	25	1	38	0.70	1.12		114											1	7	2.80									

[illegible]

New.SNo	Sno	NAME	Sub_cli	Age	Trime	GA	TSH	FT4	TPOAb	OGCT	TREAT	mat_com	PRE ECLAMPSIA	GHTN	GDM	IUGR	OLIGO	PRETERM	FETAL DISTRESS	LABOUR COMPLICATION	mod_deli	APGAR	baby_wei	fet_com	LOW APGAR	LBW	PRETERM	NICU	RDS	TFT	DELAYED PASSAGE OF MECONIUM	PROLONGED NEONATAL JAUNDICE
411		Meentchi	2	28	1	38	1.51	1.10		90		previous lscs									2	7	3.00									
412		Kreethana	2	21	3	37	1.72	1.50		96		previous lscs									2	7	2.60									
413		Palavi	2	22	2	39	1.66	1.02		89											1	7	3.00									
414		Selvi	2	25	1	38	2.01	1.08		78											1	7	2.80									
415		Jency	2	23	3	37	1.81	1.00		92		GHTN,Failed induction		2							2	7	2.75									
416		Prema	2	26	2	37	1.95	1.11		88											1	7	2.90									
417		Bhuvana	2	27	3	37	1.30	1.21		101		Previous lscs									2	7	3.00									
418		Pooja	2	29	3	36	1.10	1.03		115		severe oligo					2				2	7	2.50									
419		Pusparani	2	31	1	37	0.40	1.05		120		Oligo					2				1	7	3.00									
421		Shiana	2	22	2	38	2.12	1.17		76											1	7	2.70									
422		Dhanabagium	2	20	3	36	2.50	1.35		84		Oligo					2				2	7	2.50									
423		Valarmathi	2	24	3	37	1.90	1.14		90											1	7	2.60									
424		Ponni	2	28	2	37	1.25	1.20		102											1	7	2.90									
425		Abirami	2	27	1	37	1.33	1.01		94											1	7	2.75									
426		Vinitra	2	29	3	37	1.76	1.19		82		IUGR Oligo				2	2				2	7	2.30	LBW/NICU Admission								
427		Poomagal	2	24	3	34	1.05	1.23		76		severe eclampsia,unfavor cx	2								2	7	1.90	LBW/NICU Admission								
428		Bhavani	2	30	1	38	1.91	1.16		98		previous lscs									2	7	3.00									
429		Sameera	2	29	3	37	1.29	1.14		78											1	7	2.50									
430		Thenmozhi	2	28	2	38	0.70	1.02		104											1	7	2.90									
431		Nishanthini	2	24	2	37	1.20	1.05		92											1	7	2.70									
433		poomari	2	27	3	37	1.50	1.12		114											1	7	2.60									
434		Mageswari	2	29	1	38	1.21	1.31		96		previous lscs									2	7	3.40									
435		velankanni	2	33	3	37	1.00	1.21		89		Preclampsia,oligo	2								2	7	2.50									
436		Nagasundari	2	25	2	38	2.62	1.09		78		Fetal alarm signal									2	7	2.90	NICU Observation								
437		Pugalenth	2	24	1	39	1.25	1.01		92											3	7	3.00									
438		vasuki	2	29	1	37	1.38	1.18		105											1	7	2.80									
439		Medtila	2	27	1	38	1.56	1.10		120											1	7	2.90									
440		Porkalai	2	22	2	37	1.80	1.06		82								2			1	7	2.50									
441		stella	2	28	1	39	1.71	1.08		78											1	7	3.50									
442		Jenthbegam	2	21	1	38	1.51	1.70		84											1	7	2.90									
443		Janaki	2	23	1	40	1.66	1.03		93											1	7	3.50									
444		Rukmani	2	26	1	37	1.33	1.12		97											1	7	2.80									
445		Jancythabetha	2	30	1	38	1.17	1.20		102		previous lscs									2	7	2.80									
447		Sujatha	2	26	3	37	1.63	1.00		94		GHTN		2	2						2	7	2.50									
448		kalaiselvi	2	23	1	39	2.80	1.19		115											1	7	2.80									

New.SNo	Sno	NAME	Sub_cli	Age	Trime	GA	TSH	FT4	TPOAb	OGCT	TREAT	mat_com	PRE ECLAMPSIA	GHTN	GDM	IUGR	OLIGO	PRETERM	FETAL DISTRESS	LABOUR COMPLICATION	mod_deli	APGAR	baby_wei	fet_com	LOW APGAR	LBW	PRETERM	NICU	RDS	TFT	DELAYED PASSAGE OF MECONIUM	PROLONGED NEONATAL JAUNDICE
449		vidhyalakshmi	2	28	1	38	2.32	1.10		109											1	7	2.90									
450		vasantha	2	27	1	39	2.08	1.02		107											1	7	3.50									
451		karpagavalli	2	29	1	38	1.58	1.01		111		previous lscs									2	7	3.40	NICU								
452		Shobana	2	22	2	38	1.21	1.01		108		CPD						2			2	7	2.00									
453		Geetha	2	21	1	38	1.85	1.05		105											1	7	3.00									
454		Kalaimani	2	25	2	37	0.80	1.15		95											1	7	2.60									
456		Sumiya	2	31	1	39	1.20	1.03		79		Failed induction									2	7	3.00									
457		Prabha	2	26	1	37	1.40	1.33		112											1	7	2.70									
458		Olimathi	2	28	1	38	1.75	1.20		100		previous lscs									2	7	2.70									
459		Chitra	2	25	2	37	1.30	1.00		98		GHTN		2							2	7	2.70									
460		Yamini	2	27	1	39	1.70	1.09		89		Failure to progress									2	7	3.80									
461		Vijaya	2	26	1	38	1.10	1.05		86		previous lscs									2	7	2.90									
462		Boopathy	2	25	2	37	1.06	1.14		77											1	7	2.60									
463		Jayachitra	2	23	3	37	1.99	1.04		84		Oligo,IUGR				2	2				2	7	2.50	NICU/RDS								
464		Karthiga	2	27	1	39	1.52	1.12		90		CPD									2	7	3.20									
465		Mala	2	26	1	38	1.33	1.19		94		previous lscs									2	7	3.00									
466		Ellamathi	2	25	1	38	1.49	1.07		83		previous lscs									2	7	3.30									
467		Divya	2	25	1	38	2.12	1.02		78		previous lscs									2	7	2.90									
468		Ramya	2	25	1	38	1.60	1.08		92											1	7	2.80									
469		kalyani	2	23	2	37	1.40	1.30		88											1	7	2.70									
470		Durga	2	29	3	37	1.70	1.09		98											1	7	2.50									
471		Hemavathy	2	30	1	39	2.00	1.16		106											2	7	3.10									
472		Malliga	2	28	1	38	1.91	1.31		114											1	7	2.75									
473		Rajeshwari	2	23	1	38	1.50	1.29		120											1	7	2.80									
474		Sathya	2	21	2	37	2.40	1.03		118											1	7	2.70									
475	475	Mahalakshmi	2	22	2	37	2.60	1.11		95		MSAF,Fetal distress									2	7	2.90	NICU/RDS								
476	476	Hema	2	25	1	37	2.02	1.23		89		previous lscs							2		2	7	3.00									
478	478	Anitha	2	28	1	37	1.85	1.00		85											1	7	2.80									
479	479	Saraitha	2	23	2	37	1.39	1.19		94											1	7	2.50									
480	480	suganya	2	25	3	37	1.56	1.10		92		CPD									2	7	2.50									
481	481	Gomathy	2	23	1	37	2.40	1.02		105											1	7	3.00									
482	482	Kavitha	2	22	1	37	2.01	1.01		113											1	7	2.74									
483	483	Radhiga	2	27	1	39	1.95	1.22		110											1	7	2.90									
484	484	shanthi	2	27	1	38	1.67	1.16		92											2	7	3.10									
486	486	Kalpana	2	25	2	37	1.00	1.21		86		Rh-ve									1	7	2.60									

New.SNo	Sno	NAME	Sub_cli	Age	Trime	GA	TSH	FT4	TPOAb	OGCT	TREAT	mat_com	PRE ECLAMPSIA	GHTN	GDM	IUGR	OLIGO	PRETERM	FETAL DISTRESS	LABOUR COMPLICATION	mod_deli	APGAR	baby_wei	fet_com	LOW APGAR	LBW	PRETERM	NICU	RDS	TFT	DELAYED PASSAGE OF MECONIUM	PROLONGED NEONATAL JAUNDICE	
487	487	Suriya	2	23	1	38	0.80	1.31		75											1	7	2.70										
488	488	Rani	2	24	1	39	1.90	1.12		88		Failure to progress									2	7	2.95										
490	490	Priya	2	27	2	37	1.40	1.09		90											1	7	2.70										
491	491	Kasturi	2	23	1	38	2.10	1.05		107		previous lscs									2	7	3.00										
492	492	Megala	2	25	1	39	1.24	1.01		91											1	7	2.90										
493	493	Divya	2	30	2	37	1.39	1.25		95											1	7	2.70										
494	494	Bhavani	2	23	2	37	2.18	1.18		87											1	7	2.60										
495	495	Punitha	2	22	3	37	1.19	1.20		79		oligo					2				2	7	2.80										
496	496	Sangeetha	2	28	1	39	1.50	1.00		101											1	7	3.00										
497	497	Dilshath	2	25	2	37	1.20	1.07		95											1	7	2.50										
498	498	Nancy	2	24	2	35	1.80	1.02		105		Cord prolapse									2	7	2.20	NICU									

LIST OF ABBREVIATIONS

TSH	-	Thyroid Stimulating Hormone
T3	-	Tri iodo thyronine
T4	-	Tetra iodo thyronine
HCG	-	Human Chorionic Gonadotropin
Wks	-	Weeks
PIH	-	Pregnancy induced hypertension
IUGR	-	Intra uterine growth restriction
NICU		new born intensive care unit
EuTH		Euthyroid
SCH		Subclinical hypothyroidism
LBW	-	Low birth weight
Rx	-	Treatment
GDM	-	Gestational diabetes mellitus
HTN	-	Hypertension
TPO Ab	-	Thyroid peroxidase antibody